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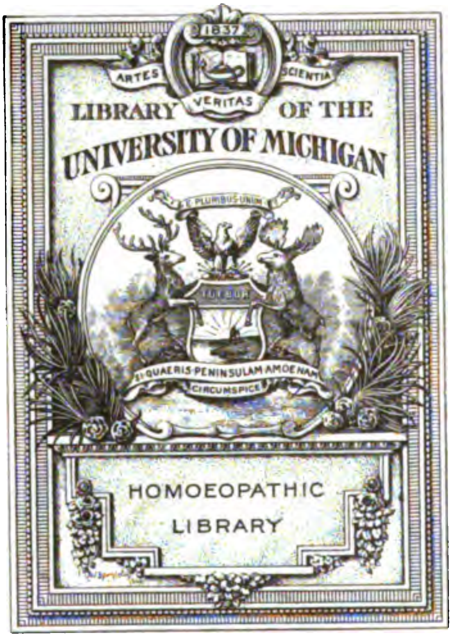

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DISEASES

of the

NERVOUS SYSTEM

By

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SECOND EDITION

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PREFACE TO FIRST EDITION

The original purpose of this book was to furnish to the students under the writer's instruction a group of lectures upon nervous diseases which would be within their powers of comprehension, and at the same time, in moderate space, provide them not only with a somewhat clear statement of neurological facts, but would also, so far as might be possible, give the reason for those statements. Having worked out this scheme, it has seemed that the general practitioner might be in a position to use such a collation to advantage, and so it is presented to the profession as an attempt to state existing facts more clearly, rather than to make any sensible addition to them. It has been the experience of the writer that books upon neurological subjects have fallen into two groups. The books of one class are so elaborate and technical that they confuse the student since he cannot construct such an elaborate picture of a disease with definiteness enough to make it possible for him to carry it in his mind. The necessary bulk of complete exposition also blinds him to the fact that he has before him *in extensio* the pathological data, and the anatomical grounds, from which he should be able to work out the typical and the unusual symptoms which have been stated in detail. The second class of books avoid this danger by their brevity, but to attain this end they are obliged to state, with the greatest economy of words, the facts of a typical example only of the disease under discussion. Statements under such limitations of space are necessarily dogmatic, and the basis for the existence of the symptoms cannot be made clear. It has seemed to the writer that, in a branch of medicine where diseases are notoriously atypical, and tend to merge into one another to all degrees, the most useful text-book was one which would make as clear as possible the pathological basis of symptoms, whenever they might occur in the nervous system, and then the character of the atypical symptoms would at once reveal the pathological lesions which must be responsible for the appearance. This is, of course, the ideal, since we do not know the actual basis of all neurological phenomena, but it has been adhered to so far as has been found practicable. The medical treatment of many nervous diseases is at present considered to be futile, so far as cure is concerned, and the physicians of all schools are driven to symptomatological prescription, and to palliatives. Under such conditions, we have a right to feel that we are the best symptomatologists yet evolved, and that by faithful work we may occasionally change the classical prognosis.

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For this reason the literature of our school has been diligently searched for remedies which are indicated, and also those giving some clinical basis for their employment. For the same reason the adjuvant and palliative agents, from all sources, have not been neglected, since the relief of suffering and deformity may be the only assistance which we can offer to many who come to us for relief. Under the conditions attending the compilation of this book, it was not feasible to denote the origin of data in the majority of instances, but the writer wishes to acknowledge his indebtedness to the many contributors of articles upon the several subjects in recent current literature, and especially to the authors of the current text-books on Diseases of the Nervous System, Therapeutics, and Electro-therapeutics. These have been freely drawn upon for material, but their presentation of the subject has been so often modified that the authors should not be held responsible for the statements in their present form.

PREFACE TO SECOND EDITION

The first edition of this book having been exhausted, the preparation of the second edition was undertaken with the knowledge that it would have to be extensively rewritten, on account of the notable extension of our knowledge of the pathology, and etiology of some diseases of the nervous system. The recent regrettable epidemics of Epidemic Cerebrospinal Meningitis, and of Anterior Poliomyelitis acuta, have given opportunity for their study under conditions absolutely unattainable previously. It has to a great degree given a new conception of their etiology, transmissability, and to some degree, of their treatment. The author wishes to acknowledge his great obligation in the preparation of these articles to the reports of the Rockefeller Institute for Medical Research, to the recent study of Meningococcus Meningitis by Heiman and Feldstein, and to the Massachusetts reports on Poliomyelitis. Recent research, all over the world, has also placed the so-called "Parasyphilitic" diseases in a new light, has altered our therapy, has somewhat bettered our present prognosis, and has given promise of a better hope for the future. Since in these diseases hypothesis has, to some degree, been replaced by certainty, it has seemed proper to extend their description much beyond their limits in the first edition. By the courtesy of Messrs. Boericke and Runyon, the publishers of the first edition, the author has been enabled to avail himself of the material in that book. He also desires to acknowledge his indebtedness, in particular among others, to the previous work of Bartlett, Dewey, Jousset and Pierce, in the department of Homœopathic Materia Medica. Recent literature in all departments has been prolific in suggestion, and it is hoped that the present work may carry out in fuller measure the intent of the earlier volume. In the preparation of the copy, and in the correction of the proof, the author wishes to record his appreciation of the very valuable assistance of Miss Gertrude Crownfield.

March 1st, 1916.
616 Madison Ave., New York

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DISEASES OF
THE NERVOUS SYSTEM

CHAPTER I.

THE ARCHITECTURE OF THE NERVOUS SYSTEM.

ANATOMY OF THE BRAIN. A detailed description of the brain and spinal cord is not properly the subject for description in a text-book of this character, but a presentation of some facts, with a different grouping, and with less detail, is necessary, in order that the significance of the symptomatology of nervous lesions may be correctly interpreted. In the center of the brain is a cavity which is enclosed by a wall of nervous tissue. This cavity is lined by a ciliated membrane, which is called the ependyma (garment), covering a layer of gray matter. This membrane is serous in character, and the cavity contains a watery fluid termed the cerebro-spinal fluid, which moderately fills it. This fluid is from 60 to 200 c.c. in quantity, increasing somewhat with age, has a specific gravity of 1.007, and contains about .01 of one per cent. of proteids, but no albumin, does not coagulate on boiling, and shows only a trace of turbidity. It contains no lymphocytes, or at the most only 2-3 to the c.m., after centrifugation, and polymorphonuclear cells are never present in normal cerebro-spinal fluid. Its pressure is from 5 up to 20 m.m. of mercury, being about that of the blood in the capillaries. The primary division of this cavity-wall is into lobes, insula, frontal, parietal, occipital, temporal, and limbic. The outer surface of these primary divisions is moulded into roll-like masses to give more space for the cell-areas. This cellular layer of gray matter is called the cortex. Internally the gray matter is in the form of laminæ and nuclei, and extended into commissures to unite the hemispheres. These external mouldings are called gyri, or convolutions; the spaces between them are fissures, which are also called sulci.

The cell areas are gray in color, while the fibres connecting them are white. The cortex is gray therefore, and the central areas are white, being made up, in the greater part, of tracts of fibres joining different cell-areas. In various places we find areas of gray matter in the central white matter; nuclei, or ganglia. These are centers where nervous stimuli are changed in direction or character of impulse. These are the caudate nucleus, lenticular nucleus, claustrum, optic thalamus, corpora quadrigemina and geniculate bodies, the amygdalæ, and the anterior and posterior perforated spaces. While appearing separate, they are connected with the gray matter of the cortex, and with each other.

There is an *unbroken continuity of gray matter* from the lamina cinerea at the front of the third ventricle to the gray surrounding the central canal of the spinal cord. The lamina cinerea is connected in front not only with the callosum, but also with the gyri of the frontal lobe, and at its sides it joins the anterior perforated space, which is the surface area of the lenticular and caudate nuclei and the claustrum, and these are connected with the gyri of the temporal lobe through the amygdalæ, which are fused with the lenticular nucleus. Keeping these facts in mind, it is easy to understand that the great ganglia are only local thickenings of the gray matter surrounding the cavity of the cerebro-spinal axis, which in the adult displays in the spinal cord the least change from the original condition.

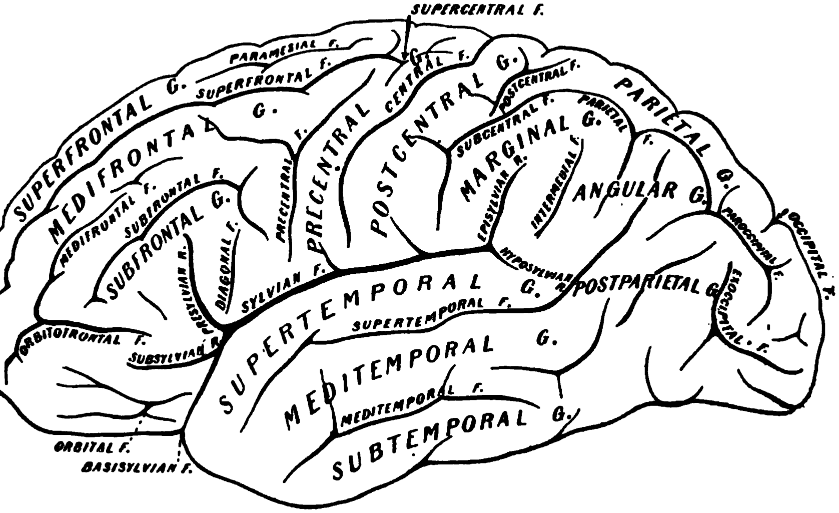


FIGURE 1. EXTERNAL SURFACE OF HEMISPHERE. (Brubaker. Courtesy of P. Blakiston's Son & Co..)

The band of white matter connecting the two hemispheres, which is called the corpus callosum, is five inches long, one inch thick behind, and a little less in front. Structurally it is a mass of nerve fibres associating the two halves of the brain. This forms the roof of the initial brain cavity, the third ventricle, from which extend the lateral ventricles at the side, and posteriorly the canal to the fourth ventricle, situated on the posterior aspect of the medulla (iter a tertio ad quartum ventriculum). From the sides of the third ventricle stretch the lateral ventricles. The anterior horn of the lateral ventricle lies in the anterior lobe of the brain, the body in the parietal lobe, the middle horn in the temporal lobe, and the posterior horn in the occipital lobe

These cavities contain certain vascular bodies called the choroid (leather) plexuses, which arise from the infolding of the pia in the process of the formation of the brain. They are the principal agents in the production of the cerebro-spinal fluid. The cerebro-spinal fluid in these cavities gains access to the serous cavities outside of the brain by a small aperture in the roof of the fourth ventricle, called the metapore, or foramen of Magendie.

The brain is covered by three membranes; the dura externally, the pia internally, and the arachnoid lying between them. The two latter are sometimes considered as one, under the name of the arachno-pia. The dura has two layers, the outer being the periosteum of the skull, and through the foramina of the skull is continuous with the pericranium. The inner layer (endocranium) is more vascular than the outer, its arteries being branches of the occipital, vertebral, ascending pharyngeal, internal maxillary, internal carotid, and ophthalmic. It is covered upon its inner surface by a layer of epithelium, which is serous, and secretes a dram or two of serum, for the lubrication of the surfaces. This is not the cerebro-spinal fluid. This membrane develops prolongations which steady the brain in the skull. The greatest of these is the falx cerebri, which is developed from the entire middle line of the vertex of the skull, and is attached to the crista galli of the ethmoid bone in front and the tentorium behind, and is the partition between the two hemispheres. The tentorium is a similar process which is arched laterally over the cerebellum. Below is the vertical falx cerebelli, and in

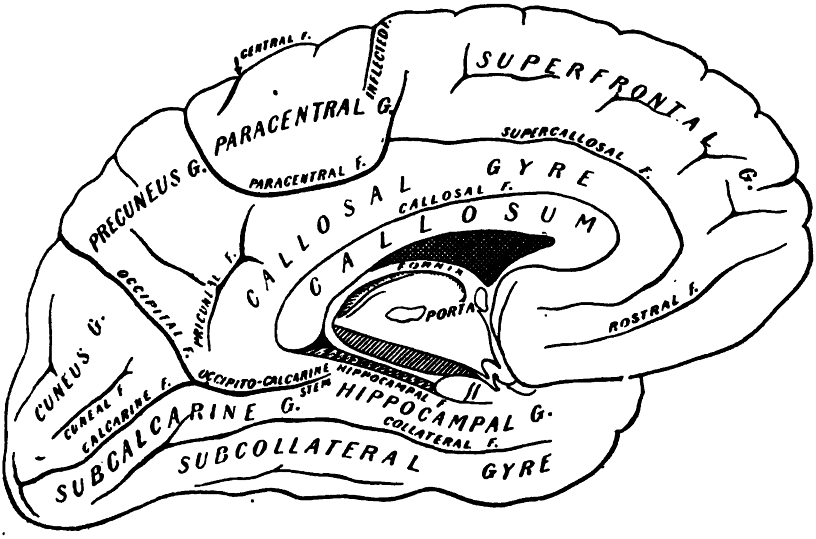


FIGURE 2. MESIAL SURFACE OF A HEMISPHERE. (Brubaker. Courtesy of P. Blakiston's Son & Co.)

front is the diaphragm of the sella, which is a small double fold of the dura extending inward from all sides over the sella turcica, covering the hypophysis, and leaving a little aperture for the apex of the infundibulum. In

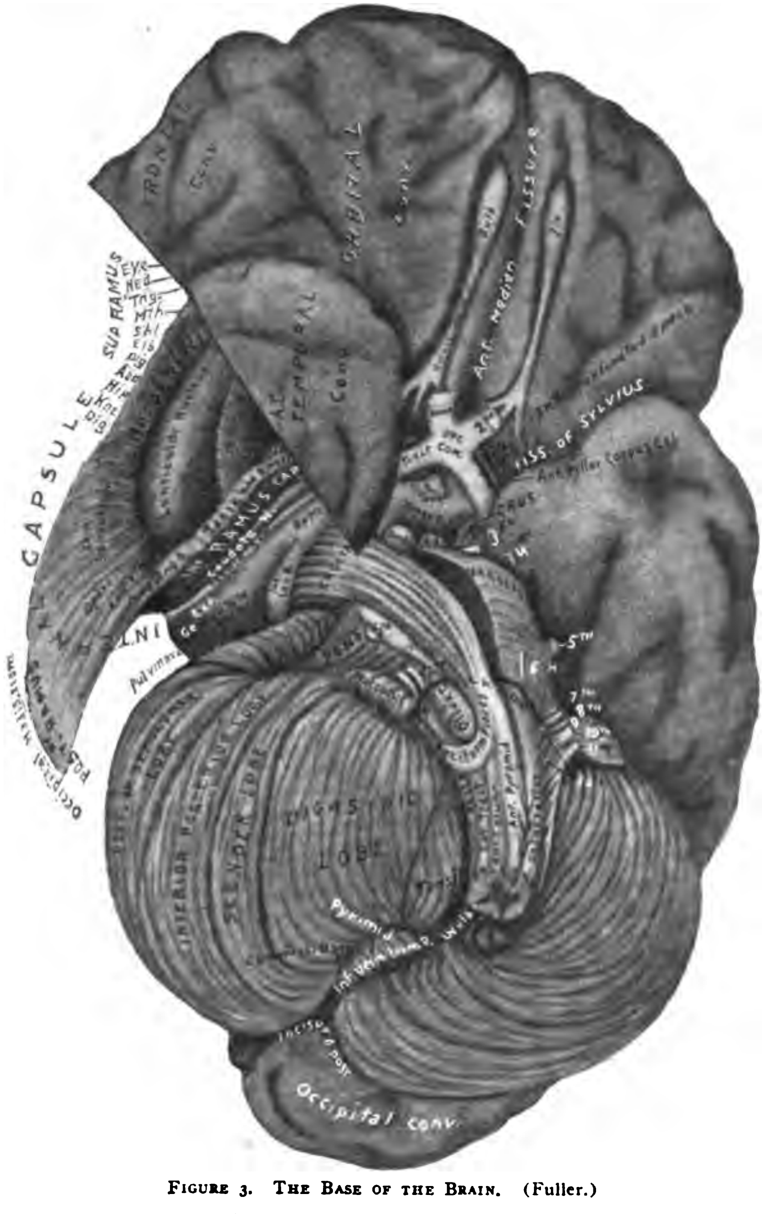


FIGURE 3. THE BASE OF THE BRAIN. (Fuller.)

the formation of the falx cerebri and falx cerebelli a triangular space is left between the two layers of the dura, and thus are formed sinuses, which are lined by an intima similar to that of the veins which empty into them. They are the recipients of the venous blood from the brain, and empty it into the general venous system, anteriorly by the ophthalmic vein into the anterior facial, and posteriorly by the veins from the occiput into the external jugular. The blood from the ventricles passes out in the veins of Galen, which empty into the straight sinus, and thence into the occipital veins, and thence into the internal jugular.

The pia is spoken of as a thin membrane which covers the surface of the brain, and follows every irregularity of its surface, but we shall gain a better idea of its function if we conceive it to be a fine meshwork of blood-vessels and lymphatics held together by a thin membrane. This blood-supply comes from the vertebrals and the internal carotid by way of the circle of Willis.

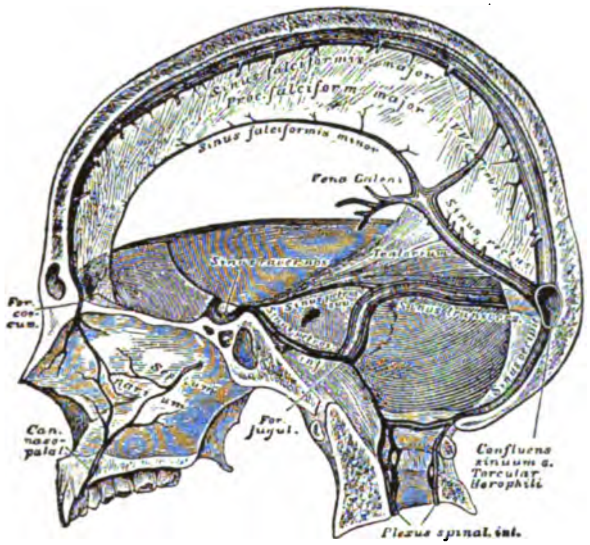


FIGURE 4. THE BLOOD-VESSELS OF THE DURA MATER. LATERAL VIEW.. (After Heitzmann.) Oppenheim. Courtesy of J. B. Lippincott Company.

Lying between this pial membrane and the inner surface of the dura is a web-like tissue, the arachnoid (spider-web). This extremely delicate structure differs from the pia in passing over the sulci of the brain instead of dipping into them, and also in that it sends off numerous partitions (trabeculæ), which divide the subarachnoid space into cavities of varying capacity. In these cavities lies that portion of the cerebro-spinal fluid which is found upon the external surface of the brain, and in health the quantity is not more than a few drams. These spaces are very much more capacious in some localities, and are then called lacunæ or cisternæ. Two large ones exist at the base of the brain, forming a water-bed for the soft brain-mass. One of these is situated between the inferior surface of the cerebellum and the dorsal surface of the medulla, while the other extends in front of the medulla, pons, interpeduncular space, and crura cerebri to the optic chiasm, and laterally to the margins of the temporal lobes. This is continuous with the anterior subarachnoid space of the cord, and above with the smaller spaces developed in front of the optic chiasm, one in each of the fossæ of Sylvius, a space over the corpus callosum, and the spaces of the medulla and cerebellum.

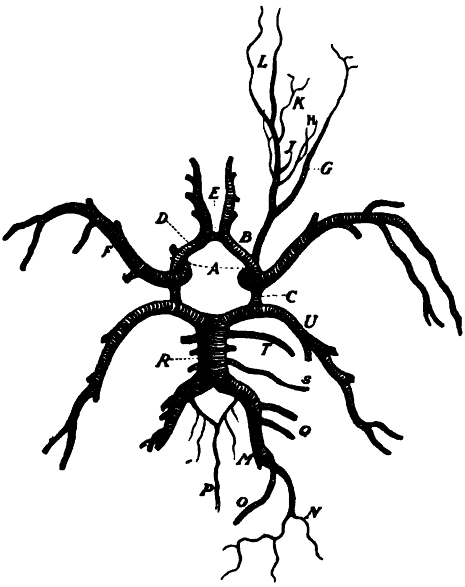


FIGURE 5. CIRCLE OF WILLIS.

A. Internal carotids. *B.* Ophthalmic. *C.* Posterior communicating. *D.* Anterior cerebral. *E.* Anterior communicating. *F.* Middle cerebral. *G.* Lachrymal. *H.* Short ciliary. *J.* Central retinal. *K.* Muscular. *L.* Frontal and nasal. *M.* Vertebrals. *N.* Posterior dural. *O.* Posterior spinal. *P.* Anterior spinal. *Q.* Inferior cerebellar. *R.* Basilar. *S.* Internal auditory. *T.* Superior cerebellar. *U* Posterior cerebral. (Modified from Bourgery.)

The blood supply to the brain comes from the internal carotid, and the vertebrals which arise, usually, from the subclavian. The perfection and certainty of supply are conserved by means of the circle of Willis. This is made up by a posterior artery of origin, which is the basilar, formed by the union of the two vertebrals. The basilar divides into the posterior cerebrals; they each send forward a prolongation, the posterior communicating, which unites with the internal carotid; the internal carotid sends forward the anterior cerebral, and the two anterior cerebrals, by their union in the anterior communicating artery, complete the circle. From this circle all portions of the brain, both external and internal, derive their arterial blood. An upward extension of the internal carotid furnishes the greater part of the blood supply to the brain; this is the middle cerebral which runs in the Sylvian fissure, and gives off the ganglionic branches to the interior of the brain. The details of supply are as follows:

The vertebrals give off the inferior cerebellar arteries, which latter give off the posterior spinal arteries; from the vertebrals then arise branches which unite to form the anterior spinal artery. They then unite to form the basilar, just at the lower anterior surface of the pons. The basilar artery gives off the transverse arteries, the median cerebellar arteries, the superior cerebellar arteries, and the posterior cerebral arteries. The vertebral arteries and their branches supply the medulla, with the nuclei of the 8th, 9th, 10th, 11th, and 12th nerves. The basilar artery and its branches supply the pons, and the nuclei of the 3rd, 4th, 5th, 6th, 7th, and 8th nerves. The cerebellum is supplied partly by the branches from the vertebral, and partly by the branches from the basilar. The tubercular quadrigemina are supplied by branches from the posterior cerebral anteriorly, and the inferior cerebellar posteriorly (Duret).

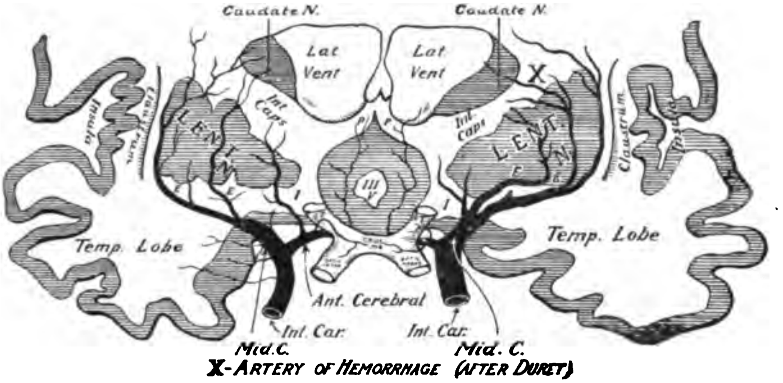


FIGURE 6. TRANSVERSE SECTION OF THE BRAIN.

These are the Lenticulo-striate arteries and are the most subject to rupture, especially upon the *left* side. Since they supply the region of the Internal Capsule their rupture naturally produces a hemiplegia, paralyzing motion and sensation on the opposite side of the body.

The posterior cerebral nourishes the internal surface of the occipital lobe, and of the temporal also, and a small portion of the outer aspect of the temporal. The anterior cerebral is distributed to the median surface of the hemisphere back to the field of the posterior cerebral on the occipital lobe, the anterior part of the outer surface of the frontal lobe, and a small strip on the external aspect of the vertex clear back to the occipital lobe. The middle cerebral supplies all the rest of the cortex, and sends off the lenticulo-striate, and lenticulo-optic branches. The former nourishes the lenticular and caudate nuclei, except the head of the latter, while the last named supplies the internal capsule, and the anterior and inner parts of the optic thalamus. The largest of the lenticulo-striate branches passes between the lenticular nucleus and the internal capsule, and is the "artery of hemorrhage" of Charcot. The finer distribution of the blood supply is principally important when it relates to the cortex. This is nourished by the blood-vessels running in the pia, from which distribution is effected by a long and a short set of branches which do not communicate, and which branches are terminal, in the sense that they have such a slight degree of anastomosis that an area of softening will result from the obliteration of any one of them. The short arterial twigs go to the gray matter, while the longer ones nourish the underlying white substance. The ganglia and nuclei have a similar supply.

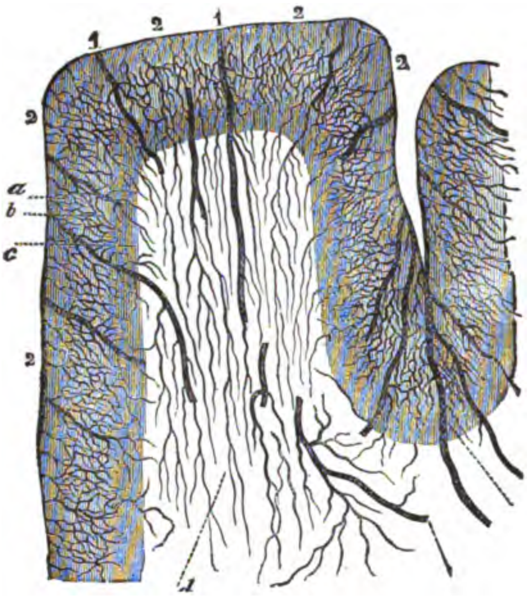


FIGURE 7. DISTRIBUTION OF ARTERIES IN THE CEREBRAL CORTEX. (After Duret.)

1, 1. Medullary arteries. 1′, 1′. In groups between the convolutions. 1″. Commissural arteries. 2, 2. Arteries of the cortex cerebri. a. Large meshed plexus in first layer. b. Closer plexus in middle layer. c. Opener plexus in the gray matter next the white substance, with its vessels. (d.) (Gordinier. Courtesy of P. Blakiston's Son & Co.)

The veins of the brain are peculiar in having no valves, in possessing a very thin wall (having no muscular coat), in emptying into the sinuses, in frequent anastomosis, in being less in number, but greater in capacity than the arteries, and in that the superior ones empty into the superior longitidinal sinus, against gravity, and against the direction of the flow in the sinus. It is to be noted here that thrombosis in the veins is made very probable from the facts just mentioned, and in the sinuses from the fact that these latter are intersected by trabeculæ. Another fact in relation to the lumen of the sinuses is that projections of the pia through minute openings in the inner wall of the dura encroach upon the lumen of the sinuses, and drain off the cerebro-spinal fluid under some conditions. These are called Pacchionian bodies, and while projecting most usually into the superior longitudinal sinus, invade other sinuses as well, and may indent the bones of the skull. The veins are divided into the superficial and deep. The superficial are the anterior and middle cerebral, and the basilar, the latter being formed by the junction of the two former. The anterior cerebral drains the median surface of the frontal lobe, and the convex surface of the corpus callosum, and unites with the middle cerebral. The middle cerebral covers a less space than the artery of the same name, lies in the fissure of Sylvius, and overlies the island of Reil, and receives the blood from this area, and unites with the anterior cerebral. The basilar runs backward from its point of origin across the crus, and around the corpora quadrigemina, and uniting with its fellow of the opposite side, and the veins of Galen from the ventricles, forms the straight sinus. The union of the two basilars in the rear, and the two anterior cerebrals in front, complete a venous circle similar to the arterial circle of Willis. The veins from the convolutions empty into the sinuses or larger veins. The deep cerebral veins drain the interior parts of the brain, and empty into the veins of Galen, which unite with the two basilars to form the straight sinus. The cerebellum has a superior and inferior system of cerebellar veins. The former empties into the veins of Galen, or the straight sinus, and the inferior into the petrosal and lateral, or the occipital sinuses.

Below the posterior part of the brain, under the tentorium, lies the cerebellum in the posterior fossa of the skull. It has no cavity, but is composed of three parts, producing a contour somewhat like that of butterfly: the vermis, which is a small lobe, but by far the most important, and a lateral lobe on either side. The whole organ is apparently made up of little leaves, the transverse fissures being so numerous and near together, and imbedded in each lateral lobe is a nucleus of irregular outline which is white within, but gray without, except at one point. The cerebellum, like the brain, has an exterior, or cortex, of gray matter, and a white central portion, striated by secondary and tertiary layers of gray matter.

Just under the cerebellum lies the medulla, which is the posterior projection of the brain, the development of the fifth vesicle of embryonic life, and downward from which extends the spinal cord.

ANATOMY OF THE SPINAL CORD.

The spinal cord is from sixteen and a half to eighteen inches in length, according to the age of the person, and reaches in the new-born to the third lumbar vertebra, and in the adult to the second lumbar, i. e., to a little below the lower border of the scapulæ. It is a half inch or less in diameter, varying at different levels, weighs about an ounce, and is always shorter, actually and relatively, in the female.

The cord is made up of thirty-one or two segments, which are the sources of the same number of pairs of spinal nerves, which are mixed in function, being composed of an anterior bundle which is motor, and a posterior which is sensory in function. These spinal nerves appear at the lateral margins of the segments. The segments are independent, each being the source of a single pair of spinal nerves, but are interdependent, since two or three segments are associated in the performance of any muscular act, or sense-perception, and the requisite coördination is ensured by the linking of the several segments to one or more, both above and below, by tracts of white fibres which run in what are called the anterior, or antero-lateral ground-bundles, which are association-tracts and also by the fibres of the column of Lissauer. The spinal nerves are level with the spinous processes in their origin and exit from the spinal canal at the upper part, but in the upper dorsai the spinous processes begin to trend downward, and the nerves do likewise, since the vertebræ are much longer than the spinal segments, so that in the lower dorsal region we must understand that the segment of origin of a nerve is a vertebra, or a vertebra and a half higher up than the point of exit of the nerve from the spinal canal. Below the ending of the cord in the conus terminale, the nerves of the lower lumbar, sacral, and coccygeal segments in their long course to their points of exit are arranged in a fan-shape, transversely in the spinal canal, called the "horse's tail" (cauda equina). From the fifth cervical to the second dorsal there is an enlargement, in order to give space for the development of the cell-areas necessary to innervate the upper extremities, and a similar, but smaller and shorter one, from the tenth dorsal to the second lumbar for the service of the lower extremities.

The cord, like the brain, is surrounded by two meningeal envelopes with the arachnoid membrane lying between them. The meninges are similar in function to those of the brain, the dura furnishing mechanical protection, the arachnoid a serous exudate, while the pia carries the vascular apparatus of the cord. There are, however, some differences which have a physiological and pathological bearing.

The spinal dura differs from the cranial in the wide separation of its two layers. The outer layer is continuous with the periosteum of the spinal canal and its ligaments, and the space between the two layers, the epidural space, is filled with a cellular structure, rich in fat, and ramified by vessels and lymphatics, rendering epidural hemorrhage much less dependent upon external violence than in the case of the cranial dura. The pia is just as closely applied to the cord as is the pia to the brain, but differs in the fact that the pia of the cord ramifies its substance with numerous septa, the folds of which form lymphatic spaces, and thus allows infections a ready entrance to all parts of the structure. This is of especial interest since Flexner and his associates have shown that some infections, poliomyelitis at any rate, gain access to the central nervous system most readily by way of the spaces about the nerves which are continuous with the spaces of the pia.

Between the dura and the pia is the arachnoid, having a slight space filled with fluid between it and the dura, i. e., subdural space, and a much greater one between it and the pia, i. e., subarachnoid space. The latter by way of the metapore (an orifice in the membrane covering the lower half of the 4th ventricle, the metatila) connects with the subarachnoid space of the brain, and thence with the cavities of the ventricles, making all these cerebro-spinal spaces into one, filled with what we know as the cerebro-spinal fluid. At the end of each recessus lateralis of the 4th ventricle there is also an opening, and each opening is called the foramen of Luschka, or of Key and Retzius.

The cord is sustained in its position vertically by the attachment of the membranes about the margin of the foramen magnum at the top, and by the fusion of the filium terminale with the periosteum of the coccyx, or of the posterior surface of the sacral canal. The filium is a fine cord made up of all the membranes fusing together, some say the dura alone, below the conus. Lateral motion in the cerebro-spinal fluid is prevented by the ligamentum denticulatum. This is a lateral band of fibrous tissue passing from the pia to the dura, running peripherally from the sides of the cord, and thus forming a partition which divides the subarachnoid space into a posterior and an anterior part. It is united to the periosteum of the vertebræ by twenty-two digitations, which are attached to the bodies of the vertebræ between the intervertebral foraminæ. By some this structure has been considered to be the avenue of drainage of the cerebro-spinal fluid.

The spinal nerves pass from the segments in an anterior and posterior bundle (actually the anteriors pass out, while the posteriors are on their way inward). On the outside of the dura, except in the case of the sacral nerves, and on the pedicle of the vertebræ there is a ganglion containing the nutrient cells of the posterior root-fibres which is called the posterior root-ganglion. Just outside of this ganglion the two nerve bundles come together.

The spinal dura and pia form the coverings for the anterior and posterior roots separately until they reach the posterior root ganglion, when they are lost in the connective tissue sheath of the nerves. Inside this sheath is the perineural lymph space communicating with the pial spaces in the cord and the lymph spaces in the epidural area.

The circulation of the cord is provided by a single artery running down its anterior surface, and by two arteries lying on the postero-lateral surface just anterior to the posterior nerves as they enter the cord. While a superficial examination would suggest that the whole cord was nourished from the vertebrals, since at the top of the cord two branches of the vertebral can be seen emerging from the foramen magnum and uniting in the anterior artery of the cord, and two other branches run about the medulla and take up a position on the cord just anterior to the posterior nerve-roots, nevertheless its blood-supply has been found to be quite segmental. For instance, if the abdominal aorta is compressed for half an hour, it is found that this cutting off the blood-supply to the lumbar and sacral vessels leads irrevocably to the necrosis of the gray matter of the lower part of the cord, and Dana points out that pathological changes in the cord are most apt to occur in the mid-dorsal region where the blood-supply from the subclavian above the heart is replaced by that from the aorta below it.

The fact seems to be that this artery, arising from a junction of the branches of the vertebrals, supplies the anterior portion of the cord only as far down as the 3rd to the 5th cervical segment. At this point it fuses with the tractus arteriosus spinalis anterior, which is made up by the union of small branches of the intercostal, lumbar and sacral arteries, which come in with every second or third nerve-root. They are five to ten in number, and are one millimeter in diameter. This anterior spinal artery runs down on the anterior surface of the cord, and not in the anterior median fissure. The posterior spinal arteries, which are given off from the inferior cerebellar branches of the vertebrals, fuse at the level of the 4th and 5th cervical nerves with the longitudinal trunks known as the tractus arteriori-postero-lateralis. These tracts are made up by the union of ascending and descending branches of the spinal rami of the vertebrals, intercostals, lumbar, ilio-lumbar, and lateral sacrals, about sixteen in number, and one-half millimeter in diameter.

The anterior spinal root arteries run into the epipial tissue opposite the anterior median fissure, and then divide into an ascending and a descending branch, thus forming that part of the anterior artery. At the level of the 9th, 10th, or 11th, thoracic nerve there is a very large root artery. The posterior spinal root arteries gain their lateral position, and break up into an ascending and a descending branch like the anterior arteries. Other branches run posteriorly, and unite in a posterior median artery. Between the postero-lateral arteries run anastomotic branches. The lower part of the cord is supplied by large root arteries from the lateral spinal arteries.

The anterior median artery thus formed runs the entire length of the cord, and throws off longitudinal branches which run in a fold of pia in the median fissure, and from these comes secondarily the supply to the anterior part of the cord. From these about 300 branches are thrown off at right angles, and one goes to one side, and the other to the opposite half, which explains the possible immunity of one-half the cord while the other half is destroyed. These branches further subdivide in the cord, so that one branch nourishes the anterior horn, and the other the neck of the posterior horn, partially supplying also the central area and the anterior portion of the posterior portion of the cord. Finally from the primary branch another division is given off vertically to unite with the branch above. The lateral roots which come in to re-enforce the postero-lateral arteries break up into plexuses, which do not seem to anastomose with the anterior median artery or the opposite postero-lateral (some assert that they do anastomose), but nevertheless the surface of the cord is covered with a rich plexus from which numerous right-angle branches penetrate and nourish the white matter of the cord. The branches of the postero-lateral arteries penetrate the cord and nourish the posterior portion, and partially the central area.

The cord has therefore a region nourished entirely from the anterior-median artery, one nourished by the postero-lateral alone, and a smaller central area with a double supply. From this condition the occlusion or rupture of a single artery is localized in its effects, although all the spinal arteries are terminal, and embolism or rupture necessitates softening. The capillaries within the gray matter empty into veins accompanying the central arteries, and these empty into veins which lie in the median fissure; most of the spinal capillaries, however, empty into fine venous twigs which make their exit on the lateral and posterior surfaces of the cord where the venous network surrounds the cord. These in turn empty into the larger veins which make their exit from the spinal canal with the spinal nerves, and these reach the vena cava while others ascend to empty into branches of the jugular vein within the skull.

The vascular system of the cord is peculiar also in the fact that it has the longest arteries of their size in the body, and that they receive the blood at low pressure, and that the veins have to act in opposition to gravity. If, therefore, the condition of the arteries is such that their elasticity is reduced, an ischemia is very likely to occur, and softening is produced. The anterior part of the cord has an abundant arterial supply, and a poorly developed venous system, so that pressure is high, and rupture is likely to occur under proper conditions, while just the opposite is true of the posterior part of the cord.

There is no true lymphatic system in the brain and cord, but the spaces between the pial envelope of the vessels and the brain tissue (circumvascular spaces) and the fissures produced by the infolding of the pia in the substance of the cord are lymph channels, and connecting with the general lymphatic system fulfil the same function. They are called the spaces of His. Since the flow from the periphery of the cord is lowest just as the perivascular lymph-spaces open into the spinal canal, the spinal roots are kept longer in contact with a possibly infected lymph than are any other structures. This probably explains the very common occurrence of root symptoms.

HISTOLOGY.

The histological elements of the nerve tissue are nerve-cells, nerve-fibres, end-organs, neuroglia-tissue, connective-tissue, blood-vessels, and lymph-channels and spaces.

The nerve unit is the neuron or neurodendron, and is made up of three parts.

1st. The nerve cell-body or neurocyte (neuron, a nerve, and kutos, a cell).

2d. The protoplasmic processes of Deiter, called also the dendrites of His, with their terminal ramifications.

3d. The so-called axis-cylinder process, with its collateral branches, and terminal end-brushes, called telodendrons.

The nerve cells, or neurocytes, are arranged in distinct groups or layers in different localities, and the areas have various shapes. In the brain there is a layer over the whole exterior aspect of the brain, and lining its cavities, while in the deep portion of the brain the cells are in small masses of various contour. In the hind-brain we find other collections, and in the cerebellum they occur like thin plates of various shape in the leaves of white tissue which make up the organ. In the crura we find the cells in narrow streaks, which continue into the pons, where again we encounter small masses. The medulla has gray matter in vertical streaks, and when we examine the cord we find that these streaks, broadened out into somewhat butterfly-like contour, have such a long vertical extent that the name "column" has been given to them. Wherever in the central nervous system we find the whitish appearance changed to a gray, we may safely assume that we are looking at a collection of cells, and if the area is a small one it is called a nucleus, but if larger and somewhat greater in bulk it is apt to be termed a ganglion, while if of long vertical extent it will be termed a column.

These cells present a large variety in shape and size in different portions of the nervous system. While originally ovoid in shape they have acquired peculiarities in form in the course of their developement. Cells are bipolar, multipolar, unipolar, pyramidal, Purkinje, and basket. Their size varies considerably, ranging from 10 to 150 micro-millimetres. The most usual shapes are irregular, large or small, multipolar forms with dendrites, or protoplasmic processes, which may be very numerous. These arise from irregularities in the surface of the cell-body, branch like a tree, but do not anastomose with one another, nor with the dendrites of other cells. They vary in length and thickness, and in their course develope frequent varicose swellings, and on their sides are minute lateral bud-like projections called gemmules. The function of the cell-body is nutritional to the entire axon, while that of the dendrites is in some way to establish contact with other cells by the way of similar dendrites given off by them. The method by which this interchange of nervous impulse is accomplished is a matter of theory, some holding that an energized cell imparts to its dendrite an erectile quality by which it is brought into physical contact with the dendrite of another cell, much as when we make an electrical contact in a bell-circuit by pushing in a button.

A rather more probable theory is advanced by Sherrington. He conceives that the dendrites and end-brushes alike are imbedded in an amorphous substance. This substance becomes energized by proper nervous stimulation, and at that moment becomes the avenue of conduction between dendrite and end-brush instead of insulating them against constant conduction and waste of nervous energy. This whole mechanism he terms a synapsis.

The typical cell is a granular protoplasmic body containing a large, usually centrally placed, nucleus, containing one or more nucleoli. This nucleus is irregular in form in the living tissue, but after death assumes a circular form. It is surrounded by a delicate membrane, and consists of a network called, from its affinity for stains, chromo-plasm, and in its meshes is a more fluid material called karyoplasm. Many of the cells of the sympathetic system contain two or more nuclei. The nerve cells of the central nervous system have no enveloping membrane, but simply a condensation of tissue.

About the nucleus of the cell, protoplasm exists in two portions: one is stainable to an intense degree, and the other a delicate coagulable substance of unknown character, which will not stain. When the stainable portion is arranged in stripes, the cell is called stichochrome, but when it is in a network the cell is termed arkyochrome. There are two kinds of stainable matter in a cell. The first is granular matter in an amorphous cement-like substance which is found in definite small collections, which will stain deeply, and are called Nissl bodies. The other stainable substance is heaped at the base of a cell in a unipolar cell, and in any case about the point of exit of the axon. In health this is a pale yellow, but in case of degeneration of the cell it stains a deep black with osmic acid, and also is found to have changed into a sort of powdery mass in place of being in granules. The non-stainable portion of a cell consists of fibrillæ, which pass in a network between the Nissl bodies, and pass into the dendrites and axis cylinders.

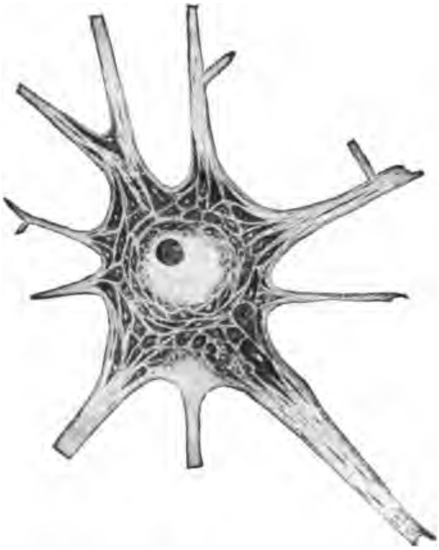


FIGURE 8. MOTOR NERVE CELL FROM VENTRAL *HORN* OF GRAY MATTER OF SPINAL CORD OF RABBIT.

Of the three lower processes, the middle one represents the axone; all the other processes are dendrites. The margin of the cells and of the masses of stainable substance appear too sharp in the reproduction. At the angle of division of the large dendrite at the left superior angle of the cell is shown one of the wedges of division. The spindle shaped Nissel bodies are well shown, especially in the dendrites. (From "The Nervous System and its Constituent Neurones," by Lewellys F. Barker. Courtesy of Wm. Wood & Co.)

Bipolar cells are found in the ganglia of the sympathetic system, posterior spinal ganglia, ganglia of the sensory cranial nerves (in embryonic life), in the molecular layer of the cortex (outside layer). They are spindle or pyriform in shape, and have a fine single axon springing from each pole.

Multipolar cells are the largest of all in the nervous system, varying from 8 to 150 micro-millimetres in diameter, which makes the largest sixteen times the size of a red blood-corpuscle, are found throughout the entire nervous system, but predominate in the anterior horns of the spinal cord, the medulla, the cerebro-spinal ganglia, and the peripheral ganglia of the sympathetic.

Purkinje's cells are found in the cortex of the cerebellum, in a distinct layer, lying between the external or molecular one, and the internal or granular layer.

Basket cells are peculiar to the cortex of the cerebellum, and they throw off axons having a horizontal course, continually giving off descending branches (collaterals) which terminate in brushes about the bodies of the cells of Purkinje. The name is evident when we see that the cells of Purkinje lie in a mass of the bushy collaterals as in a basket.

Pyramidal cells are multipolar, triangular, or pyramidal in shape, and possess a fine dendrite at the apex which tapers as it approaches the superficial layer of the cortex. Delicate dendrites project from all sides of the cell body, while from each corner of the base springs a dendrite, which extends obliquely to the plane of the vertical fibres of the cortex, and there divides into numerous branching filaments studded with gemmules. The axis-cylinder process usually springs from the middle of the basal portion of the cell-body, passes vertically downward, has great length usually, is smooth, less in diameter than the dendrites, and gives off right-angled collaterals. In most cases axons soon after leaving the cell-body become covered with a continuous layer of nucleated oblong cells, which subsequently become modified, and constitute the medullary or myelin sheath. When fresh, the myelin is clear, and semi-fluid. Its function is protection to the nerve-fibre, and possibly insulation. Fibres thus invested are termed medullated or myelinated nerve-fibres. Such medullated fibres constitute the white substance of nervous tissue throughout the nervous system, and always mean nervous paths, as opposed to cells, where some changes of a chemical nature take place upon which nervous impulses hang in some way. Stated in a dogmatic way, axons conduct sensory impulses to, and motor impulses from cell-bodies. All peripheral axons end in: (1st) inter-epithelial arborizations; (2d) specialized end-organs; (3d) motorial end-plates. The first two modes of termination appertain to sensory nerves only, while the latter refers to the combined sensory-motor nervous apparatus in voluntary muscles.

There are six chief forms of specialized sensory end-organs: (1st) The tactile corpuscles of Meissner and Wagner; (2d) the end-bulbs of Krause; (3d) Pacinian or Vater's corpuscles; (4th) tactile menisques; (5th) corpuscles of Golgi; (6th) so-called muscle-spindles. A large number of the sensory nerves do not end in these specialized methods, but in the interepithelial arborizations referred to before. In this event the axon as it approaches the skin, or the surface of the mucous membrane, loses its myelin and neurilemma, and the axis-cylinder, thus left naked, repeatedly divides into a plexus of fine fibres, which terminate in the epithelial layers of the given tissue, or the layers of other structures which it supplies. This method of termination is true of the skin, mucous membrane, hair-bulbs, teeth, tendons, the muscles, and many of the glands.

Among the specialized end-organs we find the tactile corpuscles of Meissner and Wagner where touch is most developed, viz., in the ends of the fingers and toes, less in the tip of the tongue, skin, lips, etc. These corpuscles are oval in shape, 1-3000th to 1-800th of an inch in length, and about 1-800th of an inch in thickness, composed of a spiral of nerve-filaments about, and ramifying, a mass of connective tissue.

The end-bulbs of Krause are small cylindrical or oval corpuscles, slightly bent at their base, made up of nucleated connective tissue capsules, in which there is a core of soft protoplasm, containing numerous cells, upon which the naked axis-cylinders rest. These are found in the mucous membrane of the lips, nose, mouth, conjunctiva, in the papillæ of the various extremely sensitive mucous membranes, and in the synovial membranes of the joints of the fingers, where they are called articular end-bulbs.

The Pacinian or Vater corpuscles are irregular oval-shaped bodies, 1-20th to 1-10th of an inch in length, situated on some of the cerebro-spinal sympathetic nerves, composed of laminated connective-tissue capsules, with 40 to 50 laminæ like an onion, with a soft protoplasmic core, through which the axis-cylinder, bereft of its myelin sheath, passes to nearly the extremity of the capsule. Here it ends in a sort of varicose swelling. These are found in the subcutaneous tissue on the nerves of the fingers and toes, near the joints, on the intercostal nerves, those of the arms, neck, nipple, external genitalia, and on the nerves of the abdominal sympathetic, and the mesentery.

The tactile menisques of Ranvier occur in both the superficial and deep layers of the skin, and are plexuses of nerve-fibers forming aborizations whose branches become flattened so as to resemble leaves.

The corpuscles of Golgi occur in tendons near their point of insertion into the muscles. The tendon-fibres become somewhat enlarged, and the medullated fibres, after losing their myelin, penetrate these fibres; then as an arborization break up into a number of fibrils, which form terminals somewhat spindle-shaped. The enlargement of these fibres of the tendons, together with these terminal arborizations of the nerves form the structures which are known as the corpuscles of Golgi.

Muscle-spindles are found in nearly all the skeletal muscles, but are especially abundant in the biceps of the arm, and the small muscles of the hand. They are more in the belly of the muscle than in the tendons, uncommon in the muscles of the eye, diaphragm, and the intrinsic muscles of the tongue.

These spindles vary in length from 1 mm. to 4 mm. They consist of a capsule of fibrous tissue, containing from five to twenty muscle-fibres. After penetrating the several layers of the capsule, the nerve fibres lose the neurilemma and myelin sheaths. The axons or axis-cylinders then divide into several long narrow branches which wind themselves in a spiral manner around the contained muscle-fibre, and terminate in small oval-shaped discs. Similar endings have been observed in the tendons of muscles. They are sometimes found degenerated in tabes dorsalis (locomotor ataxia). Their function is the setting up of contractions in muscles.

Motor nerves terminate in both voluntary, and involuntary muscles. In the voluntary muscles the nerves are all medullated, and have their origin in the cerebro-spinal system. The nerves to the involuntary muscles are non-medullated, and belong mostly to the sympathetic system. Motor nerves to voluntary muscles terminate chiefly in special expansions called motor nerve-plates or organs, motor-sprays, fields of innervation of Kuhne, eminences of Doyers, or sarcoglia. These end-plates are located below the sarcolemma of primitive muscle-fibre, are continuous with their sarcous substance, are flattened or slightly elevated masses of granular protoplasm, irregular, spherical, or oblong in shape, contain cells with an investing envelope, and clear nuclei, and nucleoli. Each motor-fibre passing into a muscle repeatedly divides at the nodes of Ranvier into branches both ascending and descending. These subdivide into a number of branchlets, each pursuing an oblique or transverse course between muscle-fibres forming an intra-muscular nerve-plexus. Each primitive nerve-fibril, still medullated, passes to a muscle-fibre, having divided just before reaching it. As it enters it, it loses its myelin, and neurilemma, and becomes continuous with the sarcolemma of the muscle-fibre. The axis-cylinder passes on under the sarcolemma, resting on the motorial end-plate, where it divides into two or three primary branches, which further subdivide into ultimate flat twigs which expand at their ends into minute bulbs. The nerve-termination is therefore a distinct non-anastomosing arborization, the whole resting upon a motorial end-plate, which is in turn continuous with the sarcous element of the muscle-fibre, and it is probably the cause of the contractile wave of the muscle, as its point of origin is in the motorial end-plate.

The motor nerves for involuntary muscles are non-medullated, and come mostly from the sympathetic nervous system. Near their termini they ramify, and form a close network, or plexus of fibres. In the angles formed by the crossing of the fine fibrillæ of this network, ganglion cells are found. From these ganglionic plexuses fibrillæ arise which pass between the muscle-fibres, and continue parallel with them, frequently bifurcating in their course, each division giving off small branches which terminate in varicose or bulbous extremities opposite the nuclei of muscle-cells, without passing into them. According to Arnold and others, they do pass into the muscle-cells and end in their nuclei.

The brain and the other parts of the central nervous system, i. e., the brain, bulb, and cord, are essentially simple aggregates of the cells which have been described, and their prolongations into dendrites, or axons. As in all other structures of the body, however, a framework of neutral tissue is found, which here is called neuroglia, or simply glia. This is a substance made up of cells with many hair-like filaments, which are termed glia, or spider, cells. They are in a state of potential overgrowth, so that when true nervous tissue becomes disintegrated, the glia cells spring into accelerated growth, and fill in the space left by the decay of nervous tissue. It is like scar-tissue in other parts of the body, and is the reason why organic diseases of the nervous system are so often incurable, for when the glia tissue has filled in the space, no renewal of nervous tissue is possible.

PHYSIOLOGY.

Physiology. In order to appreciate the causation of diseases of the nervous system, we must grasp the fact that each nerve-unit (neuron-cell, dendrites, axon, terminal brushes) is an easily excitable body, and that all movement and function may properly be considered as a response to peripheral or central stimuli. If the spinal centers or their axons are injured, the result will be disease of the cord as a reflex organ.

If the brain or fibres from it in the cord (pyramidal fibres) are injured the brain will lose the power of inaugurating movements of the body in response to the will or of controlling the reflex activities of the cord. On the other hand, if the sensory fibres are destroyed in the cord there will be a loss of intellectual appreciation of the significance of external stimuli.

If the terminal brush of a nerve-fibre is applied to non-nervous tissue, the axon is a motor one, and is efferent, or one conducting impulses from the center to the periphery, while the contrary is true if the terminal brush of an axon ends about nervous tissue within the cord. One end of each neuron is receptive, and the other emissive, and the cell is the nutrient center for the whole structure. The connection between terminal brushes and cells is by means of fine filaments in the dendrite which are called neuro-fibrils. The terminals of the dendrites collect impressions, and these are conveyed by the neuro-fibrils to the cell, or to the axon passing out of it. The cell receives these impressions from the dendrite by the termination (apparently) upon its surface of the neuro-fibril in little placques which are known as the "feet of Held," or "plates of Auerbach." How the interchange of stimuli takes place from the end-brush of the recipient cell which has gathered them to the dendrite of the cell to which they next pass is somewhat in doubt. With some degree of probability it has been assumed that the contact of the terminal brush and the recipient dendrite is obtained by erectility, or through the agency of an amorphous substance lying between them, the whole mechanism being then called a synapse. The function of the synapse is conjectural, and it may be a substance capable of allowing nervous impulse to pass only under certain conditions, which would not militate against a theory of continuity of nerve-fibre, or it may be a body in which the actual contact of the fibres takes place after erectility had been produced by a stimulus to the terminal. They may be capable of this erectility only when in a condition of health, and this is possibly the cause of the lack of potential energy, both mental and physical, of a tired person. Nervous energy is exceedingly slight in amount, and traverses a fibre under usual conditions in one direction only (perhaps impulse is made uni-directional by the synapse). As a wave of nervous energy flows along a nerve it does not influence similar fibres, or the structures near it, but at a synapse it stimulates whatever dendrites are imbedded in that structure, and therefore at that point it passes the impulse on to other fibres, and at that point, and at no other, may change the direction of the impulse, and also diffuse it, since one impulse may here be received by many dendrites relating to different cells. This is the law of isolated conduction of J. Müller. This diffusion depends upon the fact that every cell has numerous dendrites, and that the terminals of a cell are in contact with numerous muscle-cells. Each neuron ends in as many as thirty brushes, and a small muscle may be innervated by two hundred spinal neurons. A simple reflex is made up of a short line with few neurons and synapses; this is a simple reflex with impulses in a single direction, whereas a more complicated arrangement means many alternative directions; this latter is the reflex used in habitual movements, and specialized groups of movements, as those of the musician, and lastly, when fibres from the ideational centers are introduced, we have the highest type of nervous linkage, where operations are carried on either reflexly or by volition.

TONUS. Every structure of the body is kept in a state of functional readiness by a constant flow of rhythmic impulses (twelve to sixteen per second), the cortical cells toning up the motor cells of the cord on the opposite side of the body, while the cerebellum energizes those of the same side.

This activity of the higher cells was formerly considered to be automatic and continuous, but Brondgeest has shown that it ceases on section of the posterior roots. It is therefore reflex in origin, and the higher cells are impelled to this by the flow of sensory stimuli from the periphery.

When a movement is inaugurated, the tonus of the cell is suddenly pushed up to the point of discharge by an augmentation of the force of this rhythmical impulse.

Nervous stimulation excites motion or function by inaugurating chemical changes, as yet undefined, in the structures. Myotatic irritability is an increased readiness of chemical combination, but the mechanism is not certain.

TREMOR. Tremor is a disorder of the normal tonicity of muscles. If this is raised we have a fine tremor marking the usual impulses, and therefore the vibrations are from eight to twelve per second, while if tonicity is diminished we have a coarse tremor of five to eight per second.

All movements which we make are slightly tremulous, for the simplest possible movement consists of the fusion of at least five contractions per second, and a movement can be repeated about eleven times per second. When the cells of the motor cortex, or the pyramidal tracts, are irritated, we may have irritation-contraction, but, although this seems to be a constant tension spasm, actually it is slightly tremulous. In general, tremor may be considered an evidence of a degree of loss of nervous energy, which, if further developed, will end in paralysis. It may arise from a disease of the cortical cells, as perhaps in alcoholism, or general paresis, or from interference with the descending tracts from those cells, either in continuity, or in the synapses, or to an increase of the inhibitory action of the higher motor-cells upon the lower. Tremor does not arise from a degeneration of pure muscle, but in cases indicating such an origin there has always been a previous injury to cells. In normal old age we see it only in voluntary movement, while if the tremor is of a part at rest, it shows an abnormal degree of cell-degeneration. The tremor of paralysis agitans arises from the fact that the impulses are too slow for fusion, and it is probably the result of a pre-senile change in nerve-cells, or in the synapse. The cells of the cortex (through the fibres of the direct pyramidal tract) inhibit a too ready motor discharge of the spinal motor-cells in response to peripheral stimuli, and certain nerves inhibit certain muscles (as the vagus does the heart), and certain regions of the brain inhibit definite parts of the body. A moderate increase of inhibition (from a general psychical stimulant like fear) will produce tremor, but if more intense will result in paralysis. Chorea may produce its characteristic incoördination in this way. Disease of nerve-fibre does not generally produce tremor, but some degree of paralysis, and when tremor appears to arise from such a cause it is probably due to degeneration in the synapse. Disseminated sclerosis is a disease marked by tremor, and probably from this cause, and in general paresis the same condition is present in the cortex, and it occurs in cerebellar disease, but only when the middle lobe is affected. It is either intention (evident only on, or exaggerated by, volitional movement), or passive, when it is only present when the part is at rest, and is stilled by attempts at voluntary motion (paralysis agitans is a typical form, being constant except in sleep).

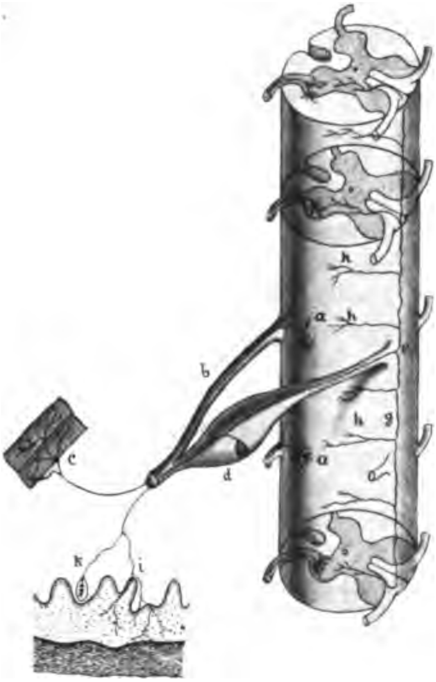


Figure 9. Scheme of a Spinal Reflex.

Cross sections of the cord are shown, with the projecting nerve roots: *c.* is a motor nerve, ending in a muscle; *k* and *i* sensory terminals in the skin and tissues; *k* is a Meissner's corpuscle, and *i* a naked arborization. They unite in the spinal nerve, *d* being the ganglion on the sensory root. *b* the motor fibre originates in a motor cell in the anterior horn, while *d*, the sensory fibre, ends in a sensory cell; *h*, the dendrite shown at *h* arborizes about one of the cells of the posterior horn. From this a new axon is sent across to arborize about one of the motor cells *a*, and thus the reflex arc is completed. (From Lenhossek.)

The essentials for reflex action are a receptive surface, an intact sensory nerve-fibre to convey the stimulus from the surface to a recipient cell, stimulation by this of an emissive (in this case motor) cell, and a responsive organ connected to the motor cell by an afferent nerve. This constitutes a simple reflex arc.

The function of the sympathetic ganglia, the cerebellum and the brain is to hold this reflex excitability under such a degree of control (inhibition) that the organism shall not be in such a state of unregulated response to stimuli continually being brought in that its energies will be exhausted in futile reactions. The reflexes which are most evident to us in health and disease are tendon reflexes (not a true reflex), skin reflexes, and the automatic functions of the bladder and rectum. It will readily be seen that if the reflex arc is broken in any part the reflex will be diminished or abolished, or if the flow of tonus from the brain is absolutely annulled, as in coma, or if inhibition is greatly increased, as in hysteria. The reflexes will be increased, on the other hand, by any condition which diminishes or cuts off inhibition, as hemiplegia, or in the functional neuroses, which increase the excitability of nerve-cells, or by degenerations of the pyramidal tracts.

Tendon-reflexes. These are commonly attributed to the propagation of a sensory stimulus by a blow, upon the patellar tendon for instance, which sends in through the sensory fibre a stimulant which irritates and discharges a motor-cell in the anterior horn of the cord, which in turn produces a contraction of the quadriceps extensor muscle. It is known to be absent in cases where there is destructive disease of the muscle, the sensory nerve, the motor nerve, or the spinal center. In spite of these lesions being sufficient to abolish this so-called reflex, it is not actually a reflex. This is proven from the fact that the reaction time is too short (.008 of a second, while nervous impulse is thirty metres per second, if it encounters no synapse) to enable the nervous impulse to travel the necessary distance. The reason that the integrity of the spinal centers and conduction-tracts is necessary is because the tonus of the muscle depends upon their condition. The reaction is a simple spasm of the quadriceps extensor varying in amplitude according as this tonus is depressed, normal, or exaggerated. Knee-jerk is always found in health, (some say absent in one per cent.), and should be single, and not reduplicated. If a stimulus is very pronounced, it may set up similar reactions in the opposite limb, while if still higher in degree the upper limbs may also be affected. This is due to the coördination of different segments by the association tracts. Other tendon-reflexes are similar. Every muscle has probably a similar jerk, but the elbow, wrist, jaw, and tendo Achillis are accessible, and so are the ones generally examined.

Pflueger has formulated a law of reflex action. Slight excitation causes a slight reflex on the side stimulated; if it is made more intense, the corresponding muscles on the opposite side of the body may be involved, yet usually to a slighter degree. If the grade of irritation is still further increased, parts higher up will join in the response through centrifugal nerves from higher segments, and if intense enough, the whole body will react, since the nervous stimulation has become so generalized as to reach the medulla.

Bladder and Rectal Control. The reflex centers for these organic reflexes are located in the fourth and fifth sacral segments, that of the bladder being below that of the rectum. The sensory stimuli arise in the mucous membrane, and the motor impulse permitting evacuation is two-fold, the tonic contraction of the sphincters is inhibited, and a contraction of the muscles of the organ is inaugurated. Owing to the fact that the somewhat complex process is under the coördinating guidance of a sympathetic ganglion the act is a very perfect reflex, and avails to empty the bladder at proper intervals, even if the path to the brain is cut off, as in the case of dorsal myelitis, but the rectum fails to functionate under the same conditions. This condition of the bladder is called active incontinence. If the sacral centers are destroyed the reflex is of course annulled, and the over-filled bladder will force open a weak or normal sphincter, and produce a dribbling discharge of a more or less constant character, this being termed passive incontinence. If the sphincter is in a state of tonic contraction, rupture of the bladder is likely to ensue. There may be a weakness of the sphincter which allows dribbling, or retention may take place from weakness of the detrusor.

Clonus is the condition where the single response of a tendon-jerk is reduplicated so long as the muscle is kept in an over-extended position, and indicates a greater degree of loss of inhibition. This is never found in functional disease, and can only arise from a degeneration of the pyramidal tracts to a greater degree than is necessary to produce a simple exaggeration of the tendon-reflexes. Ankle clonus is the one usually found, but patellar, or jaw clonus are present in rare instances. If the second and third lumbar segments are destroyed, and the pyramidal tracts are degenerated, knee-jerk will be lost, but clonus will be present.

Spasm is a contraction of the muscles exceeding the limits of health, both in intensity and duration, without volitional stimulation, or when such stimulation produces a contraction of inordinate duration. Some cases arise from a simple paralysis of opposing muscles, or some intoxicant in the blood may set up an inflammation of the fibrous sheath of the muscle, or the muscle-fibres, which, in turn, increases the irritability of the nerve-centers, but in general we may feel sure that the centers of nerve-energy are irritated, or while inhibition is weakened as one factor, the same influence has simultaneously debilitated the cells in the cord, and has made them too susceptible to peripheral stimulation.

TONIC SPASM indicates that inhibition has been weakened; if the degree is a little greater we find clonus; and if still greater, there is contracture. This is when the position assumed during spasm is retained permanently.

CLONIC SPASM is the condition when the contraction alternates for a period with relaxation, at a rate which is uniform for a single attack, but may vary in successive attacks of the same individual, or in the attacks of several individuals.

Tonic spasm can be proved to sometimes depend upon a cutting off of inhibition, since the knee-jerk will return to the tabetic if he suffers from a succeeding lesion which cuts off the pyramidal tracts, and it will occur in a tumor of the cerebellum if the growth compresses the pyramidal tracts. The cause of clonic spasm is not so certain, but conditions which produce a hemiplegia are competent for its production.

Spasm may occur in peripheral nervous diseases where the motor-fibres are irritated, but it is not universal in these conditions, and is not usually a marked symptom. In diseases of the sensory nerves it is more common to find spasm, but it is not proven that it is not the result of the debilitated condition of the spinal centers. In tetanus all the centers suffer from the intoxication, and therefore a slight peripheral stimulation tends to a sudden release of nervous stimulation to the muscles.

REFLEX SPASM sometimes occurs, but mostly in children, and in them the pyramidal tracts are poorly developed, and here it may also be based upon deficiency of inhibitory action. When a muscle is put upon a stretch by its opponents, it contracts so as to steady them. If this is continued for a length of time, the centers may fall into a state of irritable exhaustion, and spasm of the overstretched muscle may occur. Functional spasms may arise without evident disease, and while they are spoken of as functional, they probably arise from changes in the cortical cells, which result in a weakening, again, of inhibition. It is worth noting that, although they exhibit themselves as unilateral, they will be found, on close investigation, to be bilateral, and to be accompanied by more or less evident weakening of strength and of the senses, especially evident on the side of most evident spasm.

Spasm of involuntary muscles is due to irritation from local causes when in the digestive tract, and sometimes in the circulatory system, but may also be a result of cortical changes.

INVOLUNTARY TWITCHINGS arise from irritation of the motor cells when the condition is not severe enough, nor sufficiently widespread to set up a contraction of the whole muscle. The separate fibres are thrown into sudden and forcible contraction, and the balance of the muscle is destroyed. If the irritation is slight, tapping the muscle, or exposing the body to cold may be necessary to develop the twitching.

CONTRACTURE. If the irritative condition is continued for a long time, or the inhibitory control of the brain over the cord is suspended for a similar period, the limb will assume a fixed position due to one of three possible conditions: the paralysis of one group of muscles will allow the unopposed over-action of the antagonists, or the irritation of one set of muscles will overcome the normal tonicity of their opponents, or long-continued, abnormal tension of a set of muscles will induce a fibro-tendinous change in the structure of the over-strained muscles, which will be followed by contraction. In the first two instances the rigidity will *cease during sleep*, and the position may be overcome by continued force, when the contracture will suddenly give way. This is called "lead-pipe" rigidity. In the latter case pressure will reduce the contracture to some point at which the opposition to further reduction will prove irresistible.

SKIN-REFLEXES. These consist in the contraction of underlying muscles when the skin in certain localities is pricked, pinched, scratched or stroked. They are not as well understood as are the tendon-reflexes, and their centers may be in the cord, but have not been localized. They are often absent, and are not always capable of interpretation. They are usually lost when the tendon-reflexes are exaggerated, and in disease of the brain the absence on one-half the body, and their presence upon the other, points out that the side without the reflex is opposite to the lesion in the brain. Skin-reflexes are never lost in hysteria, thus again differentiating it from organic disease, but are exaggerated if the centers are irritated or inflamed, and in many functional conditions marked by diminished nervous tension they may be inhibited to a degree by volition.

NORMAL TENDON REFLEXES must be obtained when the muscle is as completely relaxed as possible, and attention must be paid to every detail of isolation of the tendon, and attitude of the patient to obtain certainty of result, which may be of very great importance for the patient's welfare, and also in medico-legal examinations.

Knee-Jerk (Patellar Tendon Reflex). Place the person in a position where the muscle can be relaxed; usually crossing one leg over another is sufficient, but it may be necessary to place the patient with the legs hanging over the edge of a table. Isolate the quadriceps tendon just below the patella, strike it a quick sharp blow with the thenar edge of the hand, or with the percussion hammer, and the result will be a prompt contraction of the quadriceps extensor. The extension of the leg upon the thigh fol-

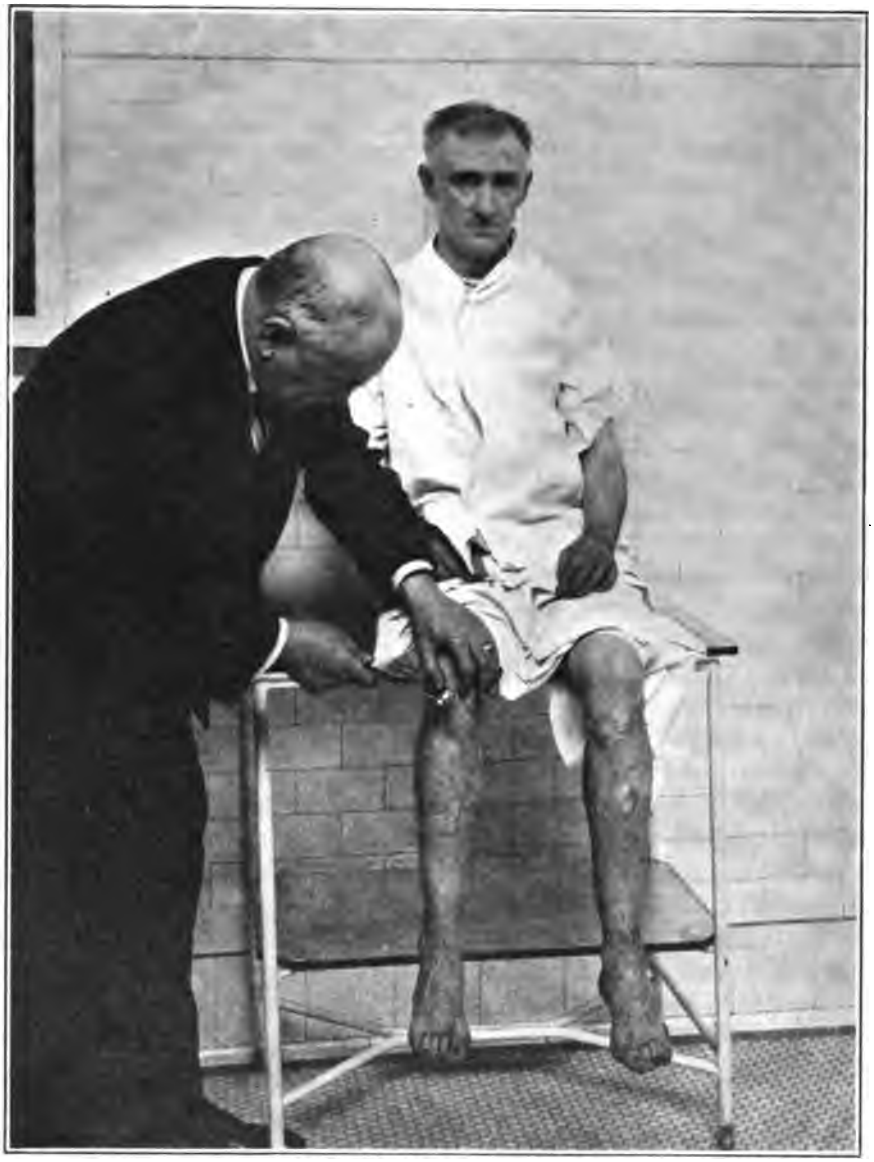


FIGURE 10. METHOD OF TESTING THE KNEE-JERK.

lows to some degree. If the patellar tendon is isolated by the thumb and finger of one hand, while the palmar surface of the hand rests upon the

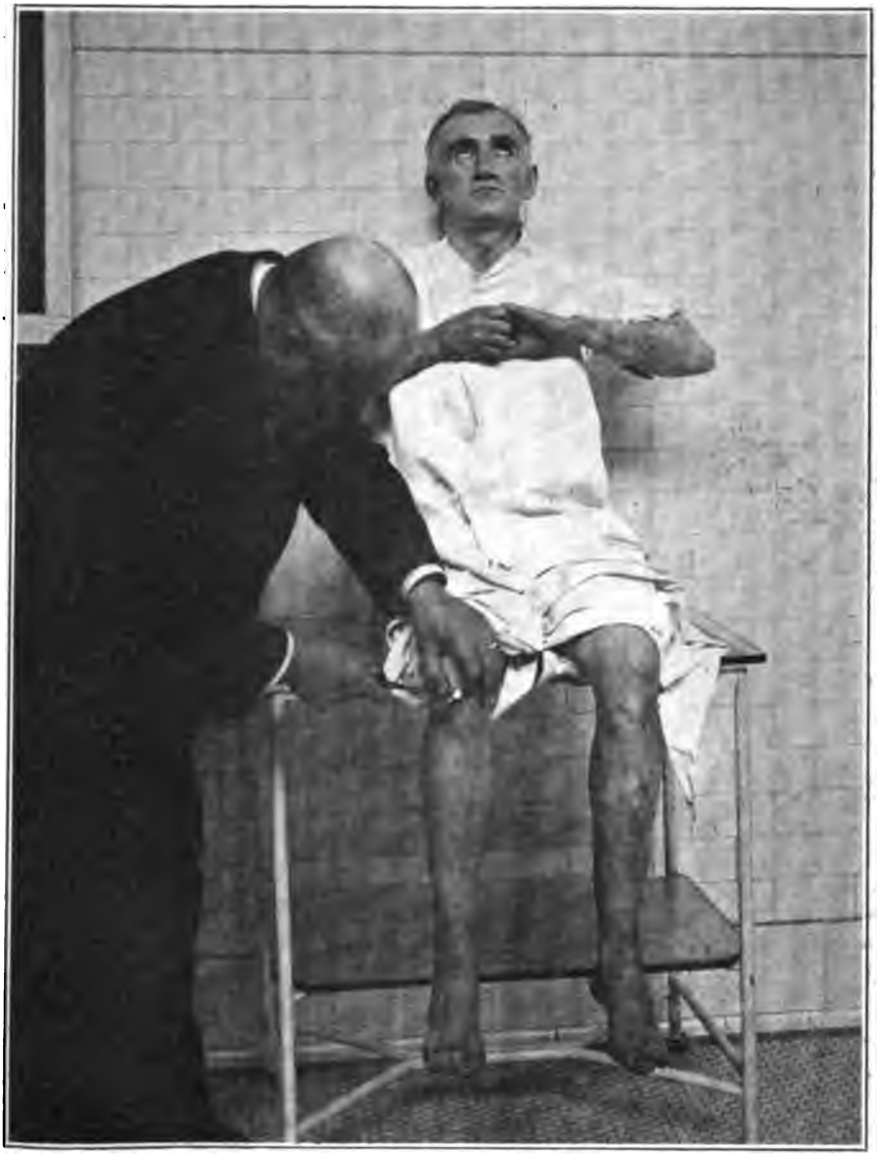


FIGURE 11. METHOD OF TESTING THE KNEE-JERK BY JENDRASSIK'S RE-ENFORCEMENT.

muscle just above the patella, a slight reflex can be discovered with certainty. The attention of the patient must be diverted where the reflex is to be carefully studied. Hooking the fingers together, and attempting to pull them apart at the word of command (Jendrassik) is a good method

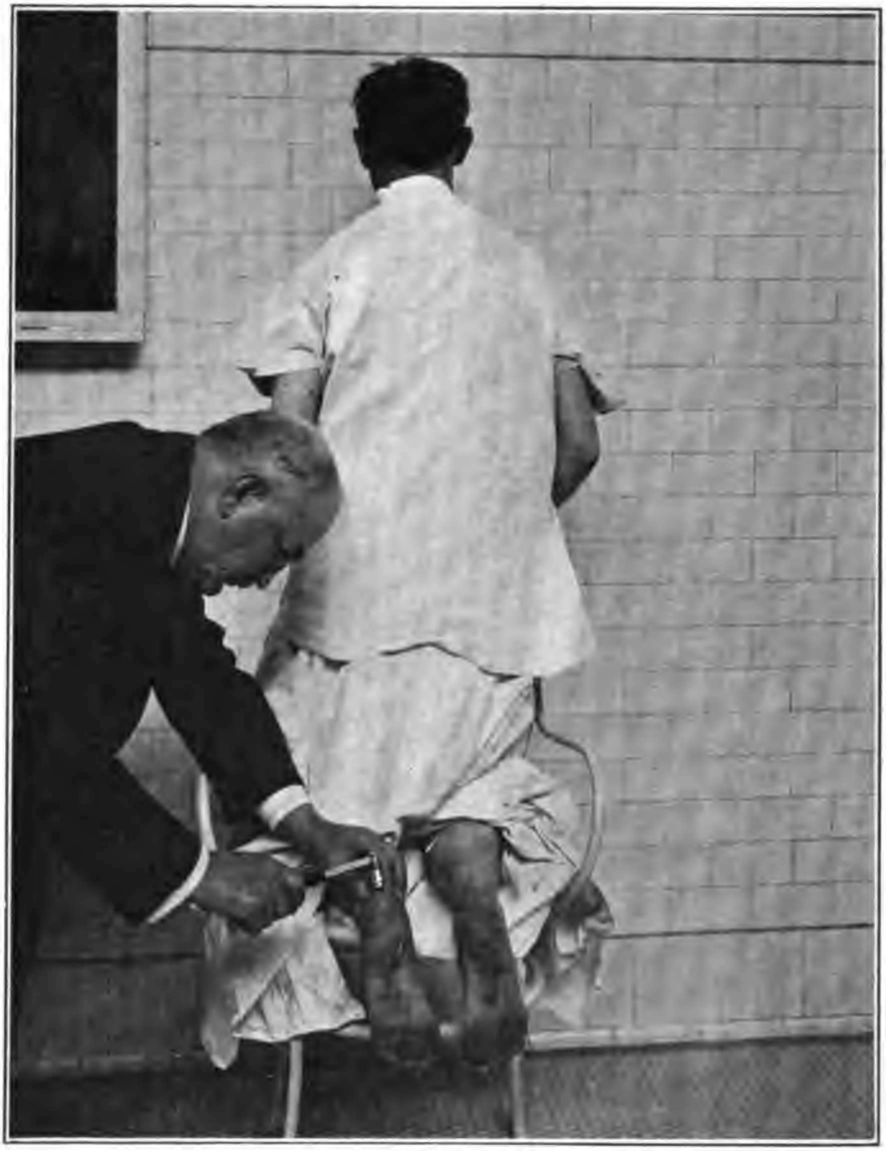


FIGURE 12. METHOD OF TESTING THE ACHILLES-JERK.

to secure this relaxation, but many other methods would commend themselves to the examiner. Allow the patient to grasp the arm of the examiner, and grip it at the word of command (Laufenauer). This reflex is present in every healthy person. (Some authorities say that it is absent in about one per cent.)

Achilles Tendon Reflex. Direct the patient to kneel on a chair, facing the back, so that the feet will project from the front edge a few inches. Sharply percuss the Achilles tendon, and the heels will be retracted.

Triceps Reflex. Support the patient's arm at the anterior surface, just above the elbow, allowing the forearm to hang loosely. Percuss the tendon of the triceps, and there will be an extension of the forearm on the arm.

Biceps Reflex. Semi-flex the forearm on the arm, and strike the tendon of the biceps, or the belly of the muscle, a sharp blow, and quick flexion of the arm will occur.

Masseter Reflex. The teeth are slightly separated, and a tongue depressor or similar object is placed in the mouth, resting on the lower teeth. A slight blow on the tongue depressor will produce a quick contraction of the masseter muscle. The same may be produced by a quick blow on the mental prominence.

Every muscle in the body has its reflex, but these are the most accessible, and they may be increased, diminished, or lost; by these differences they show the pathological condition of the nervous system.

ABNORMAL TENDON REFLEXES. Ankle Clonus. Seat the patient comfortably. Support the leg on one hand in a semi-flexed position. Force the foot dorsally, by pressing with the fingers on the sole of the foot, at the root of the toes. A to and fro motion of the foot will occur, which constitutes what is termed clonus. In order to indicate organic damage to the nervous system it must be inexhaustible, for four or five oscillations may be discovered in many neurasthenic persons. The author prefers the use of the fingers instead of the palm of the hand, in order to cause the clonus by delicate accession and relaxation of pressure, since the phenomenon may be very difficult to demonstrate.

Contra-lateral Reflex. When the patient lies on the back with the lower limbs semi-flexed, and the patellar tendon is percussed, the leg will be extended upon the thigh, and the same will occur in the opposite limb.

NORMAL CUTANEOUS REFLEXES. Stimulation of a sensory nerve may set up a muscular contraction in the area of that nerve, but if the individual is nervously diseased, the muscles of the entire body may be stimulated to contraction.

Plantar Reflex. In a person in health, slight stroking of the plantar surface of the foot will cause plantar flexion of the toes, and, according to individual susceptibility, of the other muscles above, perhaps to the extent of flexion of the leg, and the thigh upon the body.

Abdominal Reflex. If the muscles of the abdomen are stroked transversely in the upper or lower quadrant of the abdomen, there will be a contraction of the abdominal muscles of that side.

Cremasteric Reflex. If the skin of the internal aspect of the thigh is stroked (sometimes irritation is demanded) the testicle of that side suddenly ascends in the scrotum.

Anal Reflex. If the skin about the anus is irritated, there is a contraction of the sphincter.

ABNORMAL CUTANEOUS REFLEXES. Babinski's reflex, Oppenheim's reflex, and the Paradoxical Flexor Reflex of Gordon. These reflexes all consist in an extension of the great toe, and possibly of all the other toes, and sometimes of a fan-like abduction of the lesser toes, the method of production of the phenomenon, only, being different.

Babinski's phenomenon is produced by a light stroking of the sole of the foot, diagonally from the outer margin at the heel, and ending under the ball of the great toe. Its presence is not diagnostic under the age of two years.

Oppenheim produces this reflex by stroking with the handle of the percussion hammer the external aspect of the tibia, exerting some pressure upon the soft parts of the leg.

The Paradoxical Reflex of Gordon is elicited when the patient is seated, and the feet, not the legs, are placed upon a stool. The limbs are everted, and completely relaxed. The examiner places the thenar and hypothenar muscles of his hands on the anterior internal surface of the tibia, and turns his fingers upward to the middle of the calf muscles. Pressure on the latter is then produced, or the fingers may roll the calf muscles from side to side.

Other reflexes will be described in the discussion of the diseases in which they occur.

SENSORY DISTURBANCES. Just as the irritation of motor tracts produces an increase of function, and a destruction of them a paralysis (and of cells the same), so we find in the sensory sphere loss of function, hyperesthesias and paresthesias from irritation, and anesthesias from destruction. These are sensory illusions, but the irritant is mentally localized in the skin-areas to which the injured nerves were distributed, and whence the stimuli would usually come.

ATAXIA. We retain our ability to balance ourselves in an upright position, and to perform movements with accuracy, and to correctly appreciate the position of our limbs, in absence of information from vision, by means of the sensations conveyed to the central organs. These sensations from the skin are tactile impressions, while those from the joints, muscles and tendons are those of pressure and tension. If this information is deficient the person will present some grade of ataxia and incoördination, and he will also have some grade of asteriognosis. This ataxia or incoördination will be the result of any lesion of the cells or tracts relating to the appreciation or transmission of common sensation, but will be most pronounced when the fibres or cells concerned in the function of muscle and joint-sense are injured. Locally considered we shall find ataxia if there is a lesion of the parietal lobe, and it often has been found when the motor cortex has been the seat of a lesion, since over much of its area there is an admixture of sensory cells. It will follow a lesion of the posterior part of the internal capsule, and has followed a lesion of the corpora quadrigemina. A lesion in the crus which is capable of producing an oculo-motor palsy with hemianopsia will also produce some degree of ataxia. A lesion in the pons which produces anesthesia in the distribution of the fifth nerve, or a facial palsy, will induce ataxia. A lesion in the medulla which causes deafness, or a hypoglossal palsy will also produce an ataxic condition:

ATROPHY. Generally it has been held to be true that the nutrition of the various parts of the body is regulated and effected by the activity of cells lying in the anterior horn of the spinal cord. Some have deduced from post-mortem findings that this is the specialized function of a definite group of cells lying to the inside and centrally from those purely motor in function. Others are just as certain that nutrition is merely an accessory function of the motor cells in whatever portion of the anterior horn they may happen to lie. Starr says that nutrition is regulated by the cells in the gray matter of the cord, and also by the local regulation of the blood supply. It is generally true that trophic disturbances from lesions of the nerves never occur except when the sensory nerve is implicated. In any case, it is certain that any structure that is cut off from the influence of its segment of the anterior horn-cells will atrophy. If this cell-destruction is a gradual process the structures will gradually waste, but if sudden, as in inflammatory conditions, the atrophic process will be so intense that sloughing will ensue, as is seen in myelitis, hemorrhage into the cord, and less frequently after intense shocks and degenerative processes in the brain.

THE SYMPATHETIC NERVOUS SYSTEM.

ANATOMY. The sympathetic nervous system is not an independent structure, but is a somewhat specialized portion of the central nervous system. It may be divided into three divisions, viz., a chain of from twenty to twenty-three inter-connected ganglia lying upon the sides of the vertebral column; the pre-vertebral plexuses, cardiac lying above the heart and supplying it, solar lying behind the stomach and supplying it, and the abdominal viscera and their vessels, and the hypogastric which lies between the two iliac arteries, and supplies the pelvic viscera; secondary, and isolated plexuses, often of minute size, which are in or near various organs, and preside over their functions, e. g., in the heart, uterus, supra-renal bodies and intestines. Each of these ganglia is reddish gray in color, and soft in consistency, but is enclosed in a tough fibrous capsule. A ganglion is made up of nerve-cells, and medullated, and non-medullated fibres. The ganglia are connected with the spinal cells and spinal nerves by tracts which are called rami communicantes, with one another by rami internodiales, and with the subsidiary plexuses by rami peripherales. The rami communicantes are found only in connection with the thoracic, and first (and sometimes the second) lumbar nerves, the second, third and fourth sacral nerves, and possibly the third, seventh, ninth, tenth and eleventh cranial nerves. These rami communicantes are the avenues of communication between the ganglia and the spinal cord, and contain medullated fibres which are derived from the motor and sensory cells of the cord, and arborize about the cells of the connected ganglion, or one in the vicinity, in which latter contingency they pass through the first ganglion on their way to the second. They also contain non-medullated fibres which arise from the cells of the sympathetic ganglion and pass to the cord, to preside over its vegetative functions, or pass thence to their destination in the spinal nerves. The rami internodiales are simply commissures between contiguous ganglia. We find then that the appearance of the ganglia near the spine is that of oval bodies connected to the spinal cord, and also with ganglia above and below them, and sending off a separate branch of communication with the less closely related ganglia. The non-medullated fibres are, in place of myelin, covered by a thin fibrous envelope with cells at intervals lying between the envelope and the axis-cylinder. These are called the fibres of Remak, found also in the olfactory nerve, and in the embryo. The connection of the sympathetic nervous system with the brain is by means of the pneumogastric nerve.

PHYSIOLOGY. The sympathetic system is not a separate reflex center, but is so closely related structurally to the brain and cord that we must believe that its functional powers are derived from these higher centers, and are modified in quality and direction only in their passage through the ganglia. Under certain circumstances they are evidently capable of complicated reflex action, since parturition has been successfully accomplished by the action of the lumbar centers when the cord has been entirely severed above that point. Their inter-relation is shown by the fact that all the vegetative functions are seriously disturbed by conditions which entail severe shock to the brain or cord, and conversely it is as true that a perversion of the vegetative processes may disturb or totally inhibit the activity of the brain and cord. Whether this result is a direct one, or whether it is the secondary result of intoxications arising from the interference with the vegetative functions is not yet known. They are, however their powers are originated, the centers of motor, vaso-motor, secretory and (perhaps) trophic impulses. They preside over the vegetative functions of life through their action upon unstriped muscle-fibre. As an exception to this rule it may be stated that they innervate the striped muscle of the heart. Hence the viscera are under their control, the dilators of the pupil, the unstriped fibres of the lids, the erectores pilorum, and the sweat and salivary glands. They also possess inhibitory functions over various structures and secretions.

PATHOLOGY. The pathology of this system is as yet in doubt, and also the diseases resulting from lesions of these nerves. Most of the conditions supposed to flow from such a cause seem possibly to be produced from disturbance of its vaso-motor function alone, and in other cases, like glaucoma, where changes have been found in the sympathetic system, the lesion has not been a constant one: a lesion in the central nervous system has at times been competent to produce an exactly similar condition. Goitre was formerly considered to depend upon diseases of the sympathetic nervous system, but it is now known that operations upon the thyroid gland, or treatment of it, are sufficient to restore the patient to health without regard to the state of the sympathetic nerves.

ETIOLOGY OF NERVOUS DISEASES.

The causes of nervous diseases are injuries, refrigeration, poisons, toxemias, infections, and exhaustions of tissue from over-use. A neuropathic heredity is the basis of a large proportion of nervous diseases and this may arise from the vicious habits of the parents, or from hardship, or mental shock endured by them. This may be direct, or atavistic, i. e., skipping a generation. It may affect children of both sexes, or of only one sex; some diseases may be effectively transmitted from the male, in other cases from the female only. It may manifest itself in a deficient developement of the tissues, whether they be the cells, tracts, or connective tissue elements, and these deficient structures are the site of later degeneration.

PATHOLOGY OF NERVOUS DISEASES.

The forms of disease affecting the nervous system are: (1) Malformations; incomplete developement, or agenesis; defective developement, or dysgenesis; these are due to disease "in utero;" (2) Hyperemia, anemia, hemorrhage, edema, and arterial and venous disease; (3) Inflammations; (4) Degenerations and atrophy, softening, sclerosis, and gliosis; (5) Infections like tuberculosis and syphilis; (6) Tumors and parasites; (7) Nutritive and functional disorders, including disorders associated with metabolic and glandular defect, such as acromegaly, and exophthalmic goitre. Since the nervous system is composed of nerve-cells and fibres, connective tissue; neuroglia, blood-vessels and lymphatics, and the lymph-spaces of His which are peculiar to nervous tissue, any disease process will be manifested in a structural or functional change of one or several of these tissues, for no process strictly confines itself to one of them, and generally involves them all. The symptoms of disease of the brain are similar in character, in spite of differences in the pathology of the process, since they depend far more upon the localization than upon the kind of disease-process primarily causing the injury of the nerve-elements. The actual changes to which these elements are subject are few, but different localizations and combinations of lesions give rise to numerous well-marked symptom-complexes. Since the blood circulates more abundantly, and at a more rapid rate in the anterior and central parts of the cord than in the posterior area, we should expect the former areas to be more resistant to disease, and this is clinically the fact. In diseases of the cord, sensory degenerations are always systematized, but if motor they are liable to be more diffuse. The brain is often affected by functional diseases (practically always nutritional or toxic), while the cord is practically immune to such conditions, except in company with the brain. Both brain and cord are equally subjects of inflammation and injury, but the cord and its meninges are more resistant to infections than is the brain and its envelopes. As a general truth it may be stated that brain conditions are almost always vascular in origin. Intoxicants sometimes damage the cells of the cortex and the nuclei, so that there is a primary degeneration of the neuraxon, but much more often this is the secondary result of a primary vascular change. The changes of mentality and function incident to old age are generally arterio-sclerotic, but may be due to a primary disintegration of the cells from the inability of these structures to longer continue the processes of metabolism to the degree necessary to healthy function. Thrombosis is more common, and leads to more serious results in the brain. The course of the vessels in the brain favors both thrombosis and embolism. The brain is often affected by abscess, hemorrhage, and tumor, while the cord is rarely subject to such damage. When the causative condition is a developemental fault, the process which results from some exciting cause is a productive inflammation, and the imperfect structures indulge in excessive cell-proliferation, which results in a pressure-necrosis of the true nervous tissue in the vicinity. The final result is a mass of sclerosis cutting off tracts, and replacing cell-areas, or softening and absorption of the new tissue ensues with the production of a cavity. This is the pathology of syringomyelia, which is a gliomatous overgrowth from the walls of the central canal of the cord, the living cells of which are to some extent still embryonic in type, and therefore are easily stimulated into overgrowth. Multiple sclerosis, since it is so often a disease of youth and early adult life, is thought by some to be a developemental fault in the myelin sheath of the nerve-fibres. After their degeneration and

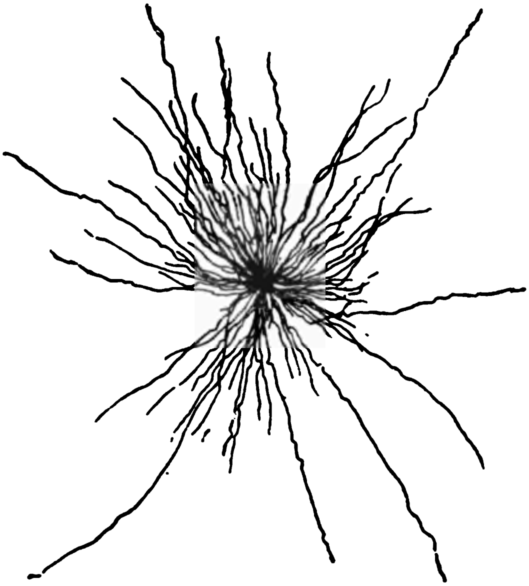


FIGURE 13. GLIA CELL.
From the spinal cord of a child nine months of age. Highly magnified. (Lenhossek.)

absorption, there is a replacement growth of neuroglia, and spots of sclerosis result which are scattered in a random manner throughout the brain and cord. The gliosis may be the primary change. Generally in this disease we find the community of structural change referred to in the introduction, since blood-vessels often show occlusion, neuroglia an irritative overgrowth, and the nerve-fibres are degenerated. Hyperemia and anemia are the cause of functional changes which may be severe and widespread in their effects. The nerve-cell is absolutely dependent upon the constant integrity and perfection of circulation, while the fibre is resistant to changes of the same degree for a long time, but cannot endure dissociation from its nutrient cell for an instant. This is probably the primary cause of degenerations in the brain and cord, although the gliomatous overgrowth clouds the post-mortem appearances. This may be simultaneous, and dependent upon the same nutritional fault. Toxins and poisons may set up the same changes, and are also nutritional, although the fault in such a case is not the volume, but the character of the blood. Edema and vascular disease destroy nervous tissue by infiltration and pressure, or by producing an ischemia, which latter produces the changes referred to above. Inflammation in any structure of the body is the tissue response to a tissue irritant, and it may be due to a chemical or toxic irritant, or to pressure. When the irritant is removed inflammation tends to subside, but since it affects not only blood-vessels and nervous tissue, but interstitial structures also, the result may be a proliferation of tissue which destroys nervous tissue which is not capable of repair, and permanent damage may be the result. The primary attack is a thickening of the coats of the arteries, and a subsequent infiltration of the surrounding tissues by an invasion of round cells. Inflammations are both exudative and productive. An exudative inflammation may be so slight that it does not produce any necrosis, or it may destroy large areas in this way. It may be purulent, and still leave the tissues capable of repair, but usually necrosis follows closely on its appearance. Most exudative inflammations are acute or subacute, and the usual forms of edema are exudative inflammations. In primary meningitis there is every evidence that the initial change is a change in the arteries, and an intense congestion. Since the periphery of the brain and the cord is nourished by short terminal arteries, if there is an occlusion of these arteries, there is a softening of the cortex of the brain, and the periphery of the cord, for a depth of half a centimetre. Myelitis is an instance of a similar condition in the substance of the cord. Here the vessels are congested, and swollen, and degenerated, they finally rupture, and leucocytes and round cells swarm throughout the tissues, and infiltrate the cells, and we find not only a primary degeneration of the nerve-fibres, but also after the myelin sheaths have been infiltrated they degenerate and expose the nerve-fibres to the direct action of the exudate, as well as to compression by it, and they break down in secondary degeneration. Productive inflammation has a chronic course with little congestion or exudation, and new connective tissue is formed from the first. Tuberculous and syphilitic processes are instances of it, and it is also caused by alcohol, lead, arsenic, and other metallic poisons; by toxins and auto-toxins, seen in gout, rheumatism, diabetes, and states of inanition. The primary change affects the blood-vessels, lymphatics, or connective tissue, and some have considered that all such changes are the results of a primary overgrowth of neuroglia, a gliosis.

Degeneration means the destruction of the parenchyma of an organ, and it may be acute or chronic, primary or secondary. If it be acute, we find softening or necrosis, due to the cutting off of the vascular supply, to direct injury, or to poisons causing inflammation. It may be followed by repair. Chronic degeneration is accompanied and followed by a connective-tissue overgrowth, and the production of a sclerotic area. It is a replacement process, and may go hand in hand with degeneration. If degeneration is primary, it is nutritional, or from a toxic poison which affects the fibre or cell directly. Secondary degeneration is that in which a fibre or cell is cut off from its center of nutrition, or there is an interruption of its vascular supply, but generally we refer to cases where the fibre has been cut off from its cell. Primary and secondary often occur in the same disease. Degenerations result from arsenic, lead, phosphorus, and other metallic poisons, the toxins and infections, from obliterative arteritis which occurs in old age, or from an inherent defect in cell-nutrition. The primary form is seen in the cord in progressive muscular atrophy, myelomalacia, tabes dorsalis, and other system diseases, while the secondary is seen in the lateral sclerosis which follows a hemiplegia, chronic myelitis, or syringomyelia. In the brain the primary degenerations are due to such changes as reduce the blood supply to the cells (like arterio-sclerosis and arteritis), and to the effects of toxins which have a selective affinity for nerve-cells, while the secondary degenerations result from such lesions as will cut off the fibres in their course from one set of cells to arborize about another, either in another part of the brain, or in their course to the cord. The selective action of poisons and toxins should be noted. For no assignable reason, particular agents affect one set of structures, and leave the others unaffected, and clinical experience is the only basis for deducing the probable cause of many intoxications. The peripheral nerves are subject to interstitial inflammations, like connective tissues in any other part of the body; they are peculiarly liable to secondary degeneration from infiltration of the fibres by toxic exudates from the membranes of the meninges of the brain and the cord, since these structures form their envelopes as they pass out from the skull and the spinal canal, and the lymph spaces of the nerves are continuous with these perineural spaces thus formed. The fibres are exposed to primary degeneration from the specific action of toxins and intoxicants circulating in the blood, which selectively act upon nervous structures at their weakest point, i. e., at the periphery, which is the portion farthest removed from the nutritive cell. Refrigeration is also very potent at this point. They are also especially liable to secondary degeneration from mechanical separation by injury. Hemorrhage acts by direct destruction of the nervous elements, and by infiltrating surrounding parts, some of such areas always being the site of necrosis, and by injurious pressure upon a still larger area, and functionally disturbs the whole brain by the sudden change of blood-pressure, producing molecular shock to the cells of the brain. The effect is greatest when the disturbance is a sudden one, the element of quantity being of secondary importance. A nerve-cell once destroyed is never regenerated, and, while nerve-fibres sometimes persist through a sclerotic patch, we have no data upon which to base an opinion that such a patch is ever absorbed, unless it be syphilitic in origin. Peripheral nerves, however, may regenerate, even after a long interval. Degeneration in a severed nerve-fibre begins in about twenty-four hours, and extends about a quarter of an inch toward the nutrient cell, while the whole peripheral portion is wasted. It has been found that in some cases nerves will show traces of damage all the way to, and also into the nutrient cell. It is probable that the coaptation of the two ends of the severed nerve never results in direct reunion, but repair is always by the more complicated process of regeneration. This is the constant tendency, since if the interval is not too great new fibrils develope from the intact central portion toward the periphery, apparently guided in direction by the cicatrized peripheral tissue. Reaching the periphery it regenerates the end apparatus. Another theory affirms that the peripheral portion shares in the regenerative activity.

THE CEREBRO-SPINAL FLUID.

The normal cerebro-spinal fluid is a brilliantly clear, watery fluid, of a specific gravity of 1.007, alkaline in reaction, contains a small percentage of chlorides, some serum globulin, a trace of cholin, and a substance which is not sugar, but which will reduce Fehling's solution. Albumen is not normally present, the fluid does not coagulate on boiling but shows only a slight trace of turbidity. It is under a pressure of about 125 m.m. of water, or 5-20 of mercury. Normally flows six to seven drops a minute.

CYTOLOGY. In the normal centrifuged fluid no microörganisms are present, two to three lymphocytes per cm., and a few endothelial plates. Polymorphonuclears are never found in a normal fluid. A lymphocyte is a large cell containing one large, deeply staining nucleus, with a ring of protoplasm about it. A polymorphonuclear leucocyte is a cell which contains a lobulated or horseshoe-shaped nucleus embedded in a mass of finely granulated material. The name comes from the fact that the nucleus in two cells is never of the same shape, and often appears multiple on account of its twisting about among the granular material in the cell.

Blood may appear on withdrawing the fluid. If it is incident to the operation it will sink to the bottom of the tube and the fluid will be clear above it, but if it has been free in the subarachnoid space it will be hemolyzed by the fluid and will be found as a granular detritus at the bottom of the tube. In cerebrospinal fever the fluid may be yellow. This arises from its admixture with blood from meningeal hemorrhages. The pressure of the fluid will be raised in all cases where there is meningitis, hydrocephalus, tumor of the brain and cord, and in anemia.

Cholin may be increased in organic nervous diseases and may afford some basis for a differentiation from functional complaints.

Albumen is increased in most acute conditions, myelitis, tabetic meningitis, cerebrospinal fever, meningeal symptoms from infections and intoxications, as lead (Gordon), in tabes and paresis.

Fibrin may form in a clot in the fluid after standing in cases of tuberculous meningitis (here in a fine thread mushroomed top and bottom), syphilitic and serous meningitis, and in sarcoma of the meninges.

The element reducing Fehling's solution is not universally agreed to be sugar. When reduced it indicates meningitis; especially tuberculous, or cerebrospinal.

An invasion of polymorphonuclear leucocytes indicates a strongly microbic infection of the meninges, and the number indicates approximately the virulence of it. Lymphocytes indicate a chronic, or subacute process, or the defense to the process indicated by the presence of the polymorphonuclears. If these decline in number while the lymphocytes increase it indicates a strong defense on the part of the organism. Polymorphonuclears are found in microbic invasions of the meninges, except in tuberculous cases. Here they are present only in the incipient stage, or if the disease runs a very rapid course. They are present in trauma of the brain and in otitic complications, and in the excited stages of paresis.

Lymphocytes are increased in chronic, or subacute forms of meningitis. It has been said that they are present in the spinal fluid of every syphilitic, either acquired or hereditary, except in the stage of chancre, but Purves Stewart (Diagnosis of Nervous Diseases, pg. 359) says that it is never present in syphilis unless the nervous system is specifically affected. Lymphocytosis is found in cerebrospinal syphilis, in the parasyphilitic diseases tabes and paresis, in the early stages of tuberculous meningitis, and in the chronic and convalescent stages of cerebrospinal fever. It is seen also in anterior poliomyelitis acuta, Landray's paralysis, herpes zoster, meningomyelitis. In meningitis of the brain and cord and in diffuse abscess lymphocytes will be found in connection with pneumococci, staphylococci, streptococci, or diplococci. Lymphocytes are absent from cases of softening and hemorrhage of the brain if they are unaccompanied by meningitis, and neuritis, if there is no myelitis.

THERAPEUTIC VALUE. In all cases of pressure, except from tumor, withdrawal of the fluid may be of value. In all forms of meningitis it will palliate, and may assist in the cure. In fractures at the base of the skull, in uremic coma, and in eclampsia it tends to relieve pressure, and may obviate convulsions. In all these cases not only is pressure relieved but the avenue is opened for the administration of serums in meningitis, and of antitoxins in tetanus.

TRACTS OF THE CORD.

The sensory nerve gains its impressions of the external world by means of the superficial inter-epithelial arborizations and end-organs just described. From some one of these collectors of external impressions the sensory nerve courses (centripetally as it is called) in the trunk of a mixed nerve, until it reaches the ganglion which lies in the pedicle of the vertebra. This posterior root-ganglion is made up of cells which are bi-polar, and each one of these nourishes one nerve-fibre from each end, the one extending from the surface to the ganglion, and the other from the ganglion to the cord. That fibre which goes to the cord enters it quite directly by the column of Lissauer (this is a commissural column, and as such is typical), or goes to form a narrow band (bandalette externe) in the column of Burdach, just central to the posterior horn. Many of the columns of the cord go directly up or down throughout its whole extent, and the fibre at the bottom may be identified with the same fibre at the top, or vice versa, but there are other columns whose fibres run but a short distance, and then bend horizontally to end about some cell in a segment, or several segments, removed from the site of its original appearance in the tract. Such fibres are commissures, as they tie together different segments, and different levels of the cord, for some associated purpose. The general vertical extent of a fibre in the column of Lissauer is 3-4 segments. From the bandelette the sensory fibres go in four directions:

1. Into the column of Burdach, which is the outer of the two columns

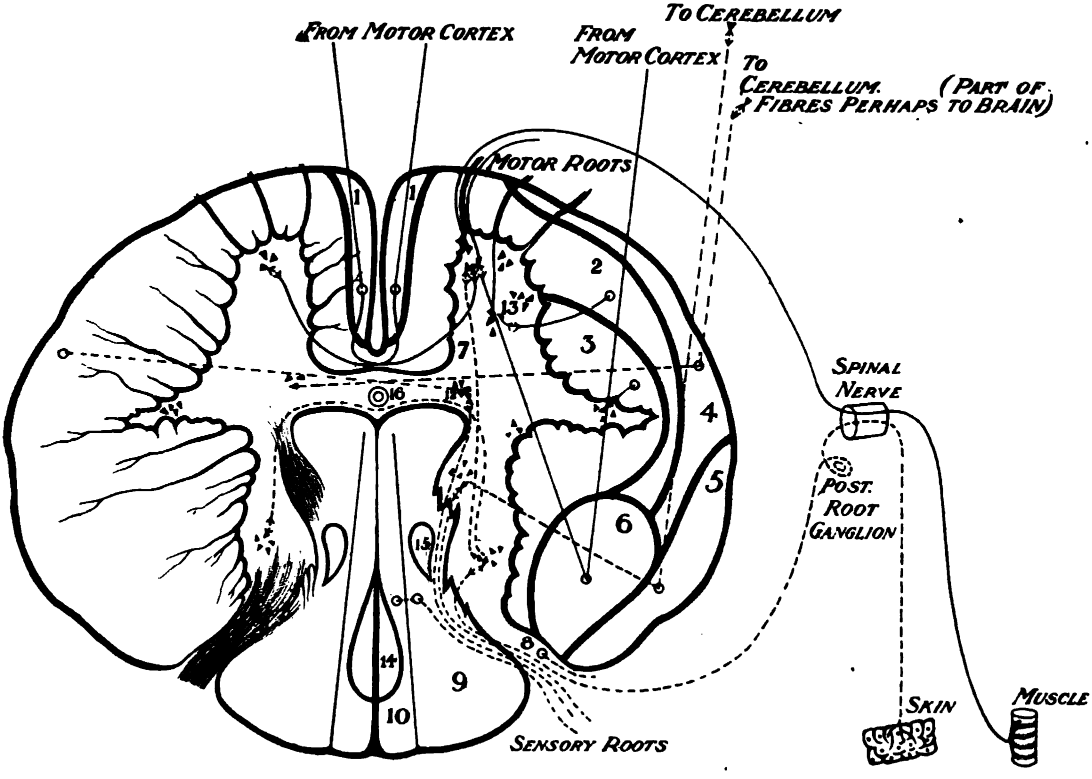


FIGURE 14. CROSS SECTION OF THE SPINAL CORD.

1. Direct Pyramidal tract (inhibitory (?) to motor cells *opposite* side). 2. Antero-lateral Ground-bundle (association tract, connecting contiguous levels of the cord). 3. Lateral Ground-bundle (association). 4. Gowers' tract (conveying to some degree impressions of pain and temperature) derived from the cells upon the *opposite* side of the cord, about the central canal (12), upward to the cerebellum and, possibly, to the brain. 5. Direct Cerebellar tract (conveying to the cerebellum impressions of pressure and tension on joints, muscles and tendons, derived from the cells of Clarke's column, unmarked, but seen at the inner aspect of the base of the posterior horns. These cells are on the *same* side of the cord as the column). 6. Crossed Pyramidal tract (conveying downward from the brain voluntary impulses to motion to the cells of the anterior horn of the same side). 7. Anterior horns (seat of motor and trophic cells). 8. Posterior nerve-roots (entrance of sensory fibres to the cord. This also covers the site of Lissauer's association tract which diffuses all sensory impressions through several adjoining segments. 9. Column of Burdach (the site of the first distribution of sensory fibres and of the change of some to a vertical direction). 10. Column of Goll (formed of fibres derived from the column of Burdach which are passing upward to the medulla and brain). 12. Cells about the central canal (recipients of sensations of pain and temperature). 13. Motor, and perhaps also trophic, cells in the anterior horn. 14. Septo-marginal tract. 15. Comma tract of Schultze. These two are descending tracts which do not degenerate with the rest of the posterior columns. 16. Central canal. When a circle is shown in a column it denotes that the fibre there changes its direction from horizontal to vertical, or vice versa.

in the space between the posterior median fissure and the posterior horn. In this column they ascend but a short distance, since the fibres constantly trend toward the median fissure, and by the time that they have ascended to the extent of a few segments they have crossed the rather indefinite line known as the postero-lateral fissure, and now are in that division of the cord called the column of Goll. New axons are continually entering the cord so that the column of Burdach is gaining from the periphery what it is losing toward the center. The column of Goll ends in the nucleus gracilis, in the lower part of the medulla, and the column of Burdach in the nucleus cuneatus. These fibres probably convey all varieties of sensation.

2. Another group of fibres turn into the posterior horn of the side of entrance, and arborize about the cells found there, while others cross in the posterior commissure to arborize about the cells of the opposite posterior horn. The axons originating in these cells situated in the posterior horn extend forward to arborize about the motor cells in the anterior portion of the cord at this level, and thus form the sensory side of the reflex arc. There are as many reflex arcs as there are muscles, but the deep reflexes principally interest us at this point. The spinal center for the plantar reflex is located in the 2nd and 3rd sacral segment. The ankle-jerk is in the 1st sacral, and 5th lumbar. The knee-jerk is in the 2nd and 3rd lumbar, and the elbow-jerk is in the 5th, 6th, and 7th cervical.

3. Another group of fibres arborize about the cells of the column of Clarke which is a column of cells lying in the anterior and internal aspect of the posterior horn, and extending from the 3rd lumbar to the 7th cervical, being most distinct up to the 8th dorsal. It has an analogue in a similar column of cells in the sacral cord called the nucleus of Stilling. From these cells new axons stream out across the posterior horn of the same side, and assume a vertical position on the periphery of the cord, just in front of the posterior roots. They then go up to the cerebellum under the name of the direct cerebellar tract. This tract extends from the 1st lumbar to the cerebellum, where it crosses and ends in the cortex of the vermis, without crossing. This tract conveys impulses from the muscles, joints, and perhaps viscera, and has to do also with coördination of movement, and especially with the maintenance of equilibrium.

4. The fibres conveying the sense of touch do not seem to have a definite path, either in the cord or medulla, some crossing in the cord, and some passing up directly. There are still other fibres which pass forward and cross to the other side of the cord in the white commissure, and finally attain a position on the antero-lateral aspect of the cord, and are known as Gowers' tract. The fibres of this tract have been assumed to arise from

secondary axons of cells about the central canal, and to convey sensation of pain and temperature, but more lately it has seemed probable that they ascend in the deep parts of the posterior horns, and of the lateral columns forming a part only of the ventro-lateral tract of Gowers. These fibres cross in part at least in the spinal cord. At the upper part of the pons many (and most of the fibres of Gowers' tract proper) curve back through the superior peduncle of the cerebellum into that organ. Other fibres, however, go through the tegmentum to the nucleus of the thalamus, and thence to the sensory motor convolutions of the cortex, and these latter are probably the conveyers of perception of pain and temperature. This tract extends the whole length of the cord. While we can divide the cord into fairly clearly defined tracts, their function is not certain, although probable, in many cases. It is probable that all the deeper sensations are carried up in the long white tracts, and the superficial ones by the short commissural fibres. These are the posterolateral and anterolateral ground bundles or association tracts, and lie between the horns of gray matter, and the white tracts on the periphery.

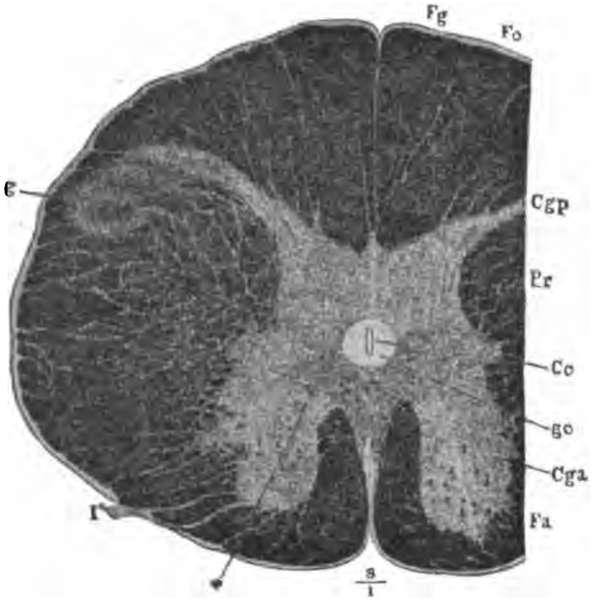


FIGURE 15. SECTION OF THE CORD AT THE LEVEL OF THE FIRST CERVICAL NERVE.

The ascending posterior column. *Fg* and *Fc* have increased greatly. The posterior columns are clubbed at the ends, and show masses of gray matter (the gray substance of Roland). *Fg* and *Fc* are about to terminate respectively in the nucleus gracilis and cuneatus on the posterior aspect of the medulla. *Cgp* is the posterior horn, and *Cga* the anterior. *Cc* is the central canal, and *gc* is the glia substance about it. (From Henle.)

The fibres are thus grouped in the cord in tracts, each one of which has a certain definite function, and when disease shows a selective affinity for one or another of these, to the exclusion of the others, we speak of the condition as a "system disease." They are then during their passage through the cord "systematized," i. e., grouped in a definite bundle, but after reaching the medulla are re-arranged. In the medulla the fibres of the column of Goll arborize about that collection of cells known as the nucleus gracilis, while the fibres of the column of Burdach end about the cells of the nucleus cuneatus. These nuclei lie on the posterior aspect of the medulla in the same relative position as the two columns hold in the cord, i. e., gracilis toward the center, and cuneatus outside of it.

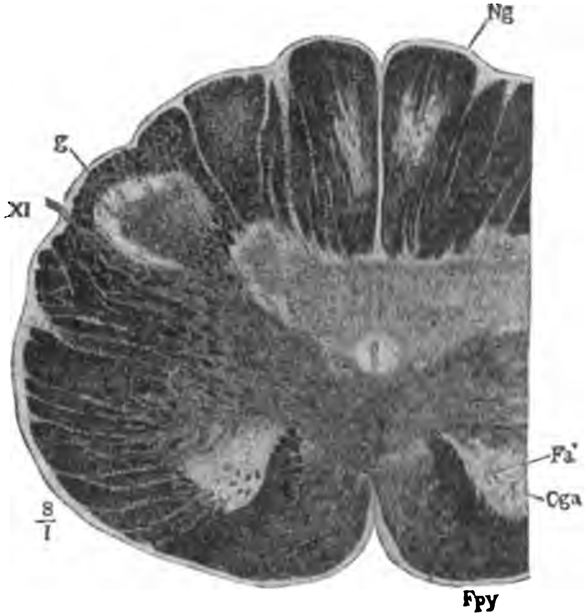


FIGURE 16. SECTION AT THE LOWER PART OF THE MEDULLA.

This is at the upper margin of the motor decussation. The fibres from the lateral tracts are seen crossing over to join the uncrossed pyramidal tracts to form the Anterior Pyramids of the Medulla. This decussation cuts off the anterior horns. At *Ng* in the column of Goll (at the top of the figure) is seen the beginning of the formation of the nucleus gracilis; *g* is the substance of Roland or gelatinosa. *XI* is the root of the eleventh nerve. (From Henle.)

At the level of the top of the cerebellar tract on each side of the medulla, and between the lateral columns and the anterior pyramids lies a large oval body, the inferior olivary body. (The superior olivary body lies in the medulla at the level of the facial nucleus and is probably the center for the re-arrangement of the fibres from the cochlea.) The sensory decussation occurs in that structure known as the formatio reticularis. The formatio reticularis is an area made up of an intricate network of interlacing nerve-fibres, interspersed with nerve-cells, extending from the medulla through the pons, and thence into the crus, where as a definite structure it dies out, since the fibres composing it are now blended with others in the internal capsule.

The fibres found are:

1. Commissural fibres of the medulla, pons, and cerebellum.
2. Cranial nerve-fibres which have a vertical direction.

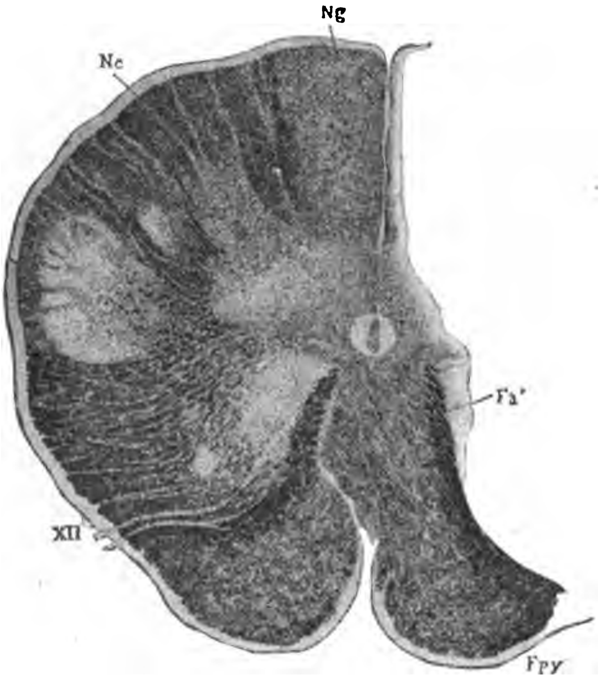


FIGURE 17. SECTION OF THE MEDULLA AT A LITTLE HIGHER LEVEL THAN THE PRECEDING.

The decussation is just beginning (if we think of the motor fibres as descending, as is the fact). The nucleus gracilis, *Ng*, occupies nearly the whole space formerly occupied by the column, and the nucleus cuneatus (from column of Burdach) also largely replaces that column. XII is the twelfth nerve arising from a nucleus which is located at the site of an anterior horn at a lower level. (From Henle.)

3. Sensory-fibres which have a longitudinal direction. In the meshes between these fibres lie isolated cells and also groups of them. These groups are:

1. Nucleus ambiguus of the 9th and 10th cranial nerves.
2. Superior olive, from which arise the 6th and 8th.
3. Nuclei of the lemniscus, a sensory group tending toward the brain.
4. Red nucleus. This lies near the aqueduct in the region of the crus, and is a nucleus into which come fibres from the anterior corpus quadri-

geminus of the opposite side, Meynert's commissure, a few fibres from Gudden's commissure, fibres from the optic thalamus, and from the lenticular nucleus, the latter fibres coming by way of the internal capsule. On the lower side, fibres enter it by way of the superior cerebellar peduncle from the cerebellar hemisphere of the opposite side.

This formatio reticularis contains a long ascending tract, as some fibres pass up through it on their way to the brain, while others come to a certain level, and then arborize about some of the cells, while still others originate in these same cells, and go thence to the brain. The tract of Gowers comes up into the formatio, but a large portion of its fibres curve over the 5th nerve, and enter the cerebellum by way of the superior cerebellar peduncle, and the velum medulare anticum, superior medullary velum, or valve of Vieussens, which is an arch of white matter binding together the lower surfaces of the two superior cerebellar peduncles. Another part of these fibres of the tract of Gowers goes upward from the formatio and terminates in part in the corpus quadrigeminus, and part in the optic thalamus,

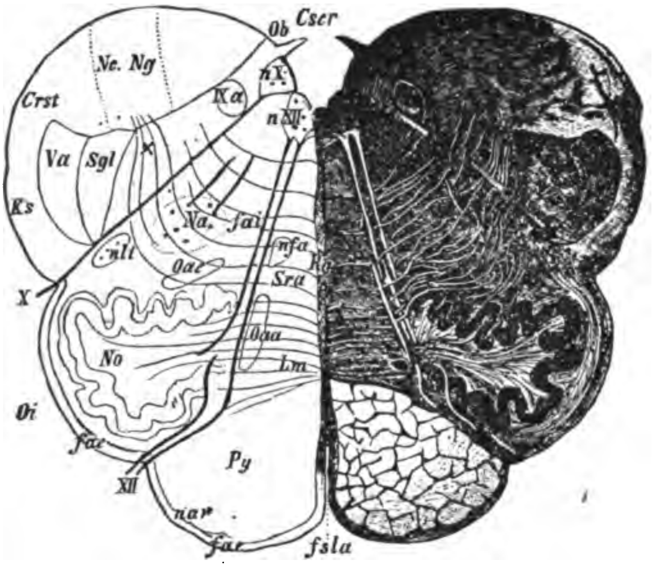


FIGURE 18. SECTION OF THE MEDULLA THROUGH THE CALAMUS SCRIPTORIUS.

The Pyramids, *Py*, have been completely formed. *No* is the inferior Olive; *Lm* is the Lemniscus; *Va* is the ascending root of the fifth nerve; *Ks* is the Direct Cerebellar tract; *Crst* is the Restiform body which becomes the Inferior Peduncle of the Cerebellum. *NX* and *NXII* are the nuclei of the tenth and twelfth nerves which nerves are seen to emerge at the margin of the figure. *IXa* (just posterior to these nuclei) is the ascending root of the ninth nerve. Tracing the fibres downward from *X* at the upper portion, we may see how the sensory decussation, or crossing, is accomplished at this point. The opening under *Cscr* (Calamus Scriptorius) is the beginning of the Fourth Ventricle. (From Obersteiner.)

the cortical representation of both groups being in the parietal lobe, the fibres passing up with those of the mesial fillet. That part of the tract of Gowers which we traced into the cerebellum arborizes there about the cells of Purkinje, from which cells axons descend in the inferior cerebellar peduncle, and penetrate the inferior olive by way of the restiform body, and thence send down fibres in the tract of Marchi and Lowenthal, the fibres from which arborize about the motor cells in the anterior horn of the same side.

By this assumed distribution of the fibres of Gowers' tract the earlier and later views of its function may be harmonized. Impressions of pain and temperature are conveyed by it to the parietal lobe (where those receptive cells are known to be located), and deep sensation to the cells of Purkinje in the cerebellum, from which tonic stimuli to the deep structures is known to proceed.

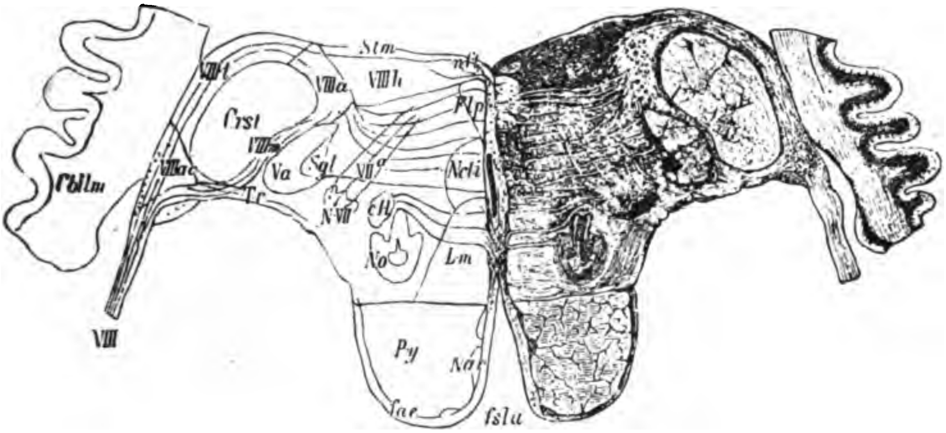


FIGURE 19. SECTION THROUGH THE MEDULLA AT THE SITE OF THE STRIAE ACOUSTICAE (SUPERIOR OLIVE).

The floor of the fourth ventricle is widened out here, and the overlying Cerebellum has been cut away, except at the sides where it appears, *Cblm*. The Restiform body is surrounded by the roots of the eighth nerve which arises from *VIII h* its chief nucleus; *VIII l* is its lateral nucleus; *VIIIm* its median nucleus; *VIIIac* its accessory nucleus *NVII* is the nucleus of the seventh nerve; *VIIa* a fibre ascending from the same; the other signs indicate parts mentioned before. (From Obersteiner.)

Ascending sensory fibres, generally speaking, gain the sensory cortex either by way of the thalamus or more directly by way of the internal capsule. In the outer third of the formatio is a collection of peculiar cells, in which end the fibres of the descending or sensory tract of the 5th nerve. These cells are situated in substantia Rolandi, which is the upward extension of a peculiar gelatinous substance, rich in cells, which is found under the same name in the posterior horns of the cord throughout their whole extent. These fibres enter the pons at about its middle, and then bend downward. The substantia Rolandi is thus seen to be the outer side of the formatio, the seat of the cell nuclei having to do with sensation for that same side in the face and head.

To resume: the main portion of the fibres arising from the nuclei gracilis and cuneatus goes in this sensory decussation (formatio reticularis), from the back of the medulla toward its front, to a position just posterior to the pyramid of that side. This bundle of sensory fibres, during this process, is called the internal arcuate bundle of fibres. On reaching its new position it at once assumes a vertical direction and proceeds toward the brain, and is now called the interolivary tract, and combined with the antero-lateral, or Gowers' tract, makes up the lemniscus (or fillet) of that side. The lemniscus comprises all the sensory fibres on the way to their distribution above.

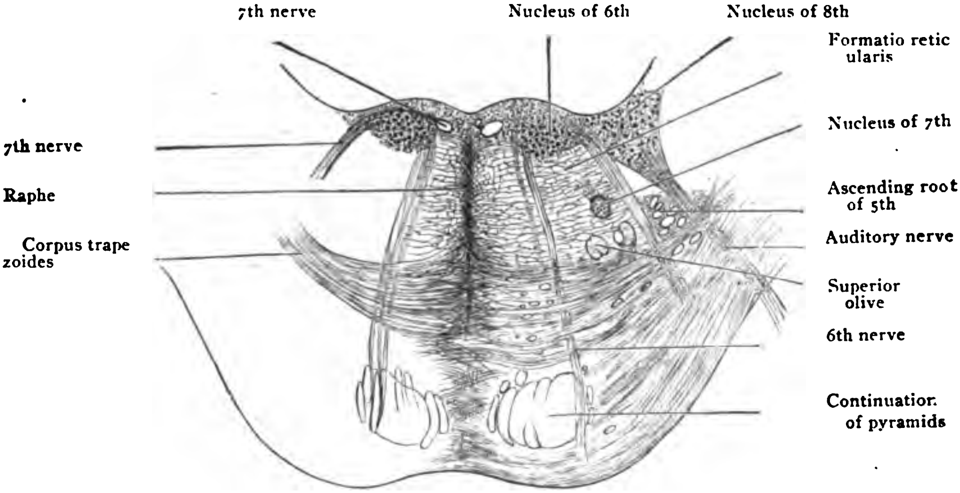


Figure 20. Transverse Section of the Pons near the Centre of the Fourth Ventricle (Schwalbe). Morris' Anatomy.—Courtesy of Blakiston & Co.

The lemniscus lies just back of the pyramids (the descending motor tracts) in the front part of the medulla. In the pons it sinks a little toward the rear, but by the time it has passed the border of the pons in its upward course, and has come to the mid-brain, or region of the crus, it trends backward, and lies beside the iter, and at the lower margin of the corpora quadrigemina, each lemniscus (here called the mesial fillet) divides into two parts. The major part after passing through the posterior corpus quadrigeminus goes to the parietal and post-central cortex by way of the optic thalamus, and the other to the same ultimate destination, but more directly, by way of the internal capsule, occupying the posterior third of the posterior limb.

We have traced the major portion of the fibres arising from the nucleus gracilis and cuneatus to their final distribution on the parietal cortex, but we must also remember that in the structure of the medulla, where they decussate, they are also known as the internal arcuate fibres.

Two other smaller bundles leave these nuclei. The first passes around the *posterior* periphery of the medulla and dips into the olive of the *same* side, which is equivalent to saying that it joins the restiform body of the same side, which is prolonged into the cerebellum as the inferior cerebellar peduncle. This bundle is one of the two groups known as posterior external arcuate fibres.

The second group from the same source, after crossing in the raphe, formatio reticularis, passes forward about the periphery of the pyramids, and enters the olive of the *opposite* side, and so joins the restiform body of that side. These are known as the anterior external arcuate fibres.

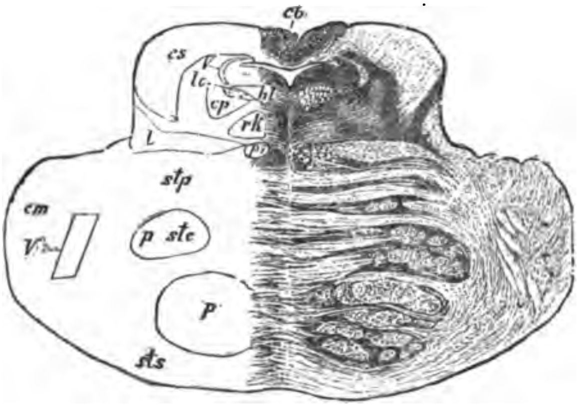


Figure 21. Section through the Pons.

(Wernicke X2) sts, stc, stp, Superficial and deep transverse bundles of the pons, intersecting the longitudinal bundles, and terminating in the mass of gray matter of the basal portion; p, pyramidal (motor) tract from the middle third of the pes; pl, median fasciculus of the pes, here seen to join the lemniscus, l; V, fifth nerve root; cm, middle peduncle of cerebellum; cs, superior peduncle of the cerebellum with cb, valve of Vieussens; lc, locus ceruleus. (Courtesy of Wm. Wood & Co.)

Summary of the arcuate fibres:

1. Internal. The majority of the fibres from the nucleus gracilis and cuneatus, which cross directly to a position back of the pyramids of the opposite side.

2. Posterior external. This goes about the posterior periphery of the medulla to the olive of the *same* side.

3. Anterior external. Cross in the raphe, and then go about the external anterior surface of the pyramids, and enter the olive of the *opposite* side.

The direct cerebellar tract, as has been said, ends in the nucleus called the inferior olive, and from the cells of this nucleus new axons extend in the restiform bodies in the inferior cerebellar peduncles, and end in the superior vermis or worm of the cerebellum. It is well, in passing, to note that the inferior cerebellar peduncle contains also the two bundles of fibres from the nucleus gracilis and cuneatus which we have just described under the name of the posterior external and anterior external arcuate fibres; also small bundles of fibres from the lateral nuclei, which are composed of cell groups from the anterior horns with some local enlargement; also the descending cerebellar tracts which are axons of the cells of Purkinje, and form the other part of the reflex arc with the columns of Gowers (these are the columns of Marchi and Lowenthal); also the direct cerebellar sensory tract from Deiter's nucleus in the medulla, where are gathered impressions from the vestibule (which tells us of the body-balance, or its position in space). These fibres cross to the other hemisphere of the cerebellum. Lastly, a tract of fibres carrying impressions outward, known as the cerebello-olivary tract, which are axons of the cells of Purkinje of the *same* side, and end about the cells of the *opposite* olive.

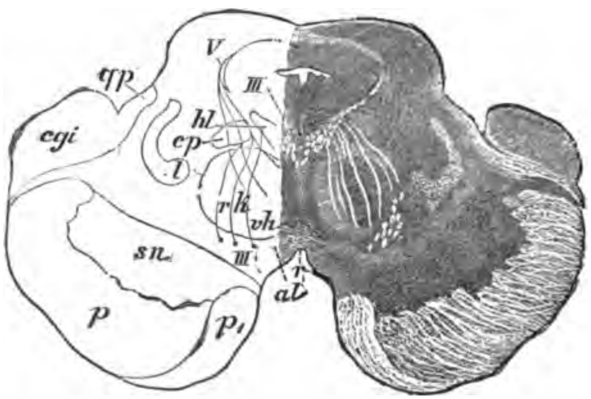


Figure 22. Section through the Crura Cerebri.

(Wernicke, X2) p, Pes peduncli; pl, median fasciculus; sn, substantia nigra; cgi, corpus geniculatum internum; qp, corpus quadrigeminum posterior; l, lemniscus; rk, red nucleus of tegmentum through which pass fibres of oculomotor nerve, III, from its nucleus in the gray matter below the aqueduct of Sylvius, to its exit between the crura; cp, fibres in the formatio reticularis which have come from the posterior commissure; hl, posterior longitudinal bundle; V, descending root of the trigeminal nerve; vh, upper part of the decussation of the superior peduncles of the cerebellum, which end in the rk; r, raphe; al, lenticular loop. (Courtesy of Wm. Wood & Co.)

The fibres in the fillet directly or indirectly arborize about the cells of the sensory areas of the brain-cortex (especially about the cells of the ascending parietal, and many cells mingled with these in what has formerly been considered a purely motor region of the cortex), and these cells send off new axons which arborize about the large pyramidal or motor cells in

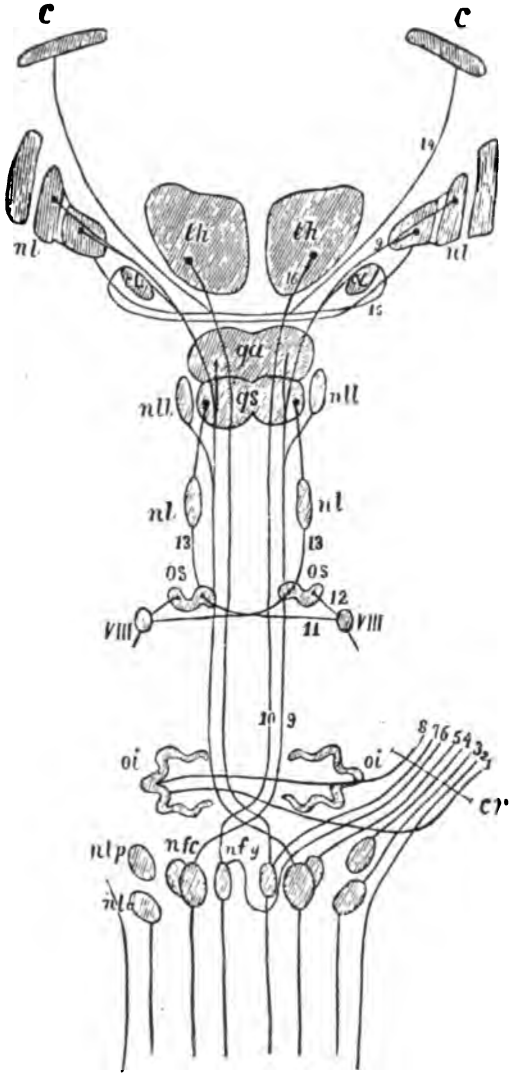


Figure 23. A Schematic Representation of the Course of the Fibres from the Nuclei of the Posterior Columns of the Spinal Cord to and through both Lemnisci, and to the Cerebellum.

C, the cortex; nl, the lenticular nucleus; th, the optic thalamus; cL, Luys' body or corpus subthalamicum; qa, and qs, the corpora quadrigemina; nll, nucleus in the lateral lemniscus; os, the superior olive; VIII, anterior acoustic nerve nucleus; oi, the olivary body; nla, nucleus of antero-lateral column; nlp, nucleus of posterolateral column; nfc, nucleus cuneatus at the top of the column of Burdach; nfg, nucleus gracilis or nucleus of the column of Goll; cr, restiform body, becoming the inferior peduncle of the cerebellum; the fibres in the latter can be traced in the figure, and need no explanation except to say that fibre 2 represents the direct cerebellar column of the spinal cord. The course of fibres from the nucleus of the lateral columns is not given. They go to the formation reticularis, and ultimately reach the parietal cortex. (From Bechterew.)

the third layer of the ascending, frontal, or precentral lobe. These cells send off in a downward direction long axons which are the longest nerve-fibres in the body, and which ultimately end in arborizations about the motor cells of the cranial nerve nuclei in the crus, pons, and medulla, and of the anterior horn at the various levels of the cord.

These descending motor fibres from the cortex of one-half of the brain are assembled at first in three fan-like bundles, which are gradually condensed into one, and turned in a quarter-circle, when they appear as a flat band of nerve-fibres lying between the lenticular nucleus on the outside, and the optic thalamus posteriorly, and to the inside, and the head of the caudate nucleus anteriorly, and also toward the inside of it. When it has gained this new position between the nuclei it is termed the internal capsule, but while in the process it has a sort of fan shape, and it then is termed the corona radiata. The point at which the internal capsule bends about the apex of the lenticular nucleus is called the knee, and the extremities are called the anterior and posterior limbs. The anterior limb, except a very small portion next the knee, is occupied by fibres which coördinate the action of the frontal lobes, and also accommodates a bundle of fibres which run from the frontal lobe of the one side to the *opposite* lobe of the cerebellum. The extreme posterior portion of the posterior of the limb seems to be devoted to a similar bundle from the occipital lobes.

Anterior to this posterior cerebellar bundle is a small area occupied by sensory fibres, both general and specialized, on their way to the cortex. The portion which is left, viz., a very small portion of the posterior part of the anterior limb, and the anterior two-thirds of the posterior limb, is devoted to the transmission of the motor fibres to the whole body, for it must be remembered that not only the motor fibres which descend in the cord are to be found here, but also those which furnish motor impulses to the cranial nerves and nerves of special sense as well.

These fibres have a definite arrangement in the capsule. Beginning a little in front of the knee and going backward, the order is as follows: eyes open, eyes turned, mouth opened, head turned (these two areas are just at the knees), tongue, mouth retracted, shoulder, elbow, wrist, fingers, thumb, trunk, hip, ankle, knee, big toe, toes.

From this comparatively wide expansion the fibres are gathered into a still more compact bundle and descend into the mid-brain, occupying that portion known as the crus. These motor fibres lie in the middle part of the crus, having on their *inner* side that bundle of fibres which was said to run from the frontal to the other side of the cerebellum, and on the *outer* side is a similar bundle running from the temporo-occipital lobe to the opposite half of the cerebellum. These compacted motor fibres are now for the first time known as the pyramids. The pyramid now enters the pons, and the fibres are again re-arranged into separate small bundles which lie on the anterior portion of the pons, and are separated from each other, as they descend vertically, by the horizontal bundles making up the middle cerebellar peduncle. On reaching the medulla, all the fibres of a side are again assembled into a compact bundle, now called the anterior pyramid of the medulla. They lie at the front margin of the medulla, but are capped toward the center of the medulla by the arcuate nucleus, and

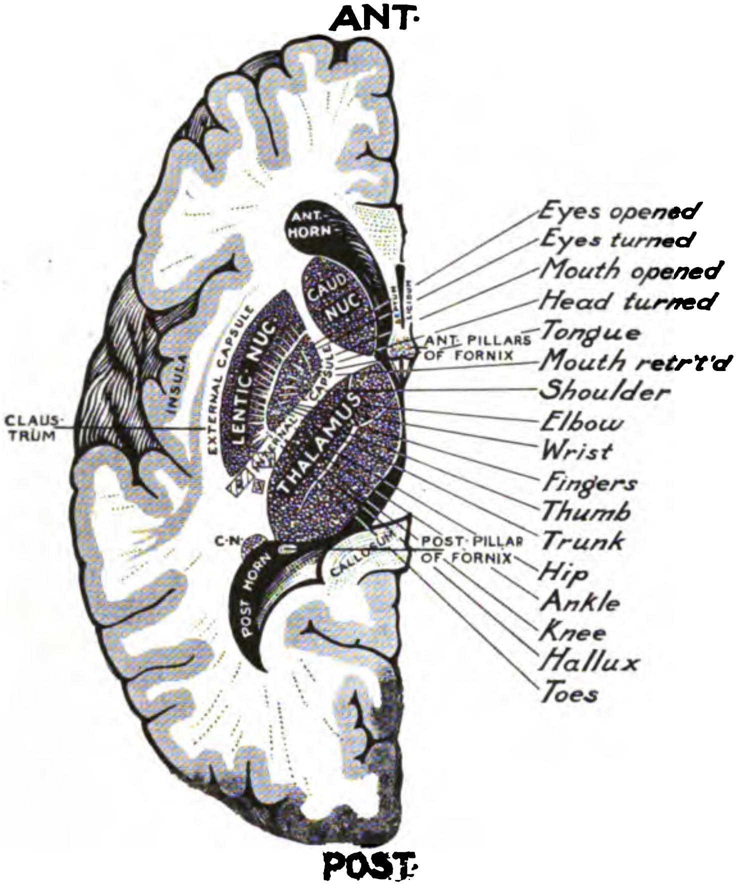


FIGURE 24. THE INTERNAL CAPSULE.

The localization of the motor divisions is indicated by the text. The small square just posterior to the "toe" area indicates the location of the tract conveying ordinary sensory stimuli, the one directly behind that is the Optic tract, and the one lying toward the median line from this is the Auditory tract. (Gerrish modified.)

separated only by the anterior median fissure. Just behind them lies the fillet or lemniscus; behind that the inferior olives. This is its position throughout the medulla, but just as it enters the cord, at the level of the *1st cervical,* it is divided into two unequal bundles, generally in the proportion of one to nine. The smaller portion, now known as the direct pyramidal tract, or the column of Turck, retains its present position throughout its whole extent, which is to about the middorsal region, rarely to 1st lumbar. It continually diminishes in size, since it continually sends off its fibres at right angles. These fibres pass through the white or anterior commissure, and arborize about the cells of the *opposite* anterior horn, and are supposed to have an inhibitory influence over the action of these cells. The other nine-tenths cross over, partially anterior, but mostly internal, to the direct cerebellar tracts near the upper part of the posterior horns. This tract is known as the crossed pyramidal tract. This crossing is known as the *motor decussation.* This column extends the whole length of the cord, but it continually diminishes, since it, as well as the direct pyramid, continually sends off fibres at right angles, which arborize about the motor cells in the anterior horn of the *same* side. These fibres convey cerebral tonus, and volitional motor impulses to these anterior horn cells. If these crossed and uncrossed pyramidal tracts are degenerated, and they are usually affected in common, spasticity below the point of the lesion is the result, on account of the unopposed cerebellar and peripheral stimuli (Dana).

The automatic control of equilibrium and muscular tonus is accomplished by what is practically a reflex arc of a higher and more complex type than that previously described. The fibres exerting this control all run in the antero-lateral ground-bundle of the side of origin, so that it may be said that the cerebral hemisphere is in communication with the opposite half of the body, while the cerebellar hemisphere affects the same side.

Afferent fibres to the cerebellum come from various sources, those by way of the superior peduncle from the red nucleus; those by way of the middle peduncle from the nucleus pontis of the opposite side, having crossed in the raphe. Those from the inferior peduncle are the most numerous and important, and come from three sources: (1) The direct cerebellar tract, which after decussation terminates in the superior vermis (or worm); (2) the anterior and posterior arcuate fibres, the former being fibres from the posterior columns of the cord by way of the nuclei cuneatus and gracilis of the opposite side, while the posterior arcuate fibres are from the nuclei of the same side. Both go to the superior vermis. (3) The acoustico-cerebellar tract composed of fibres which are the axons of the sensory end-nuclei, Deiter's, and the superior olive of the vestibular portion of the auditory nerve. It is probable that all their fibres decussate before their final distribution.

The direct cerebellar tract is found in the restiform body to be accompanied by a tract which degenerates downward. This is the column of Marchi and Löwenthal, or the anterior marginal fasciculus. It is peculiar in that it brings the spinal cord directly under the influence of the cerebellum without the mediation of any relay station. It is a narrow band occupying the border of the cord between the anterior end of Gowers' tract and the anterior median fissure. It originates in the roof nucleus of the cerebellum (nucleus fastigii), and terminates about the cells of the anterior horn of the same side.

The rubo-spinal tract is a wedge-shaped bundle, originating in cells of the red nucleus (lying in the tegmentum, just below the aqueduct); the fibres cross in the medulla, and run down as far as the lumbar cord.

The vestibulo-spinal tract arises from the cells of Deiter's nucleus (lying in the floor of the fourth ventricle), and the course and termination is the same as the rubo-spinal.

The tecto-spinal originates in the cells of the corpora quadrigemina, and has the same course and termination as the foregoing, except that it divides, sending a median tract in the anterior column, and a lateral tract in the lateral column.

These four tracts are the paths by which the cerebellum produces automatic movements, and maintains equilibrium, and the tone of the muscles.

In the posterior columns are endogenous fibres which arise from the cells of the posterior horns. Just behind the posterior commissure is one of these, the cornu-commissural tract. It is an ascending column. In the cervical region there is one situated in the median line, midway antero-posterior of the posterior columns, and is called the comma tract of Schultze.

A small bundle is found in the thoracic region at the periphery, which is Hoche's bundle.

In the lumbar region, and on each side of the median septum, and in the middle is Flechsig's oval bundle.

In the sacral region on the posterior and internal region Gombault's and Phillipe's triangle are probably the same system of fibres, and have a descending course.

The motor cells of the anterior horns send out axons which go out at the periphery of the cord singly, not leaving the cord in a compact bundle like that with which the posterior root enters the cord, and these fibres on the outside are gathered into a cord which unites with the sensory fibres just beyond the posterior root-ganglion, and at the point where it passes out of the inter-vertebral foramen which is hollowed out between the

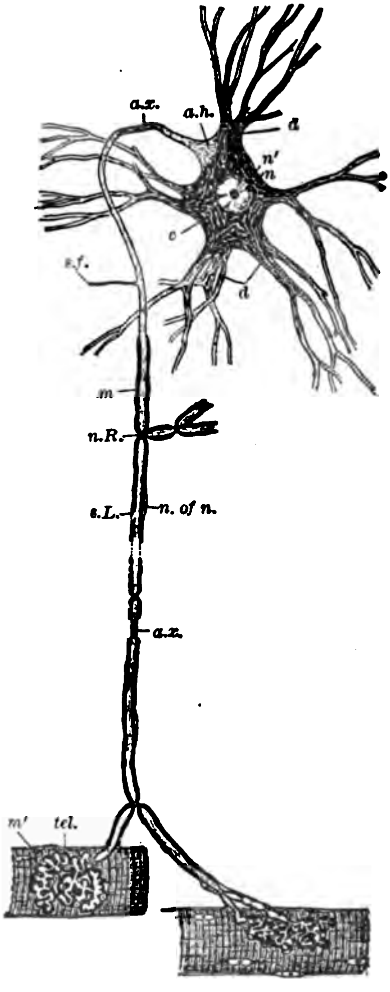


FIGURE 25. SCHEMATIC REPRESENTATION OF A LOWER MOTOR NEURON FROM THE VENTRAL HORN OF THE SPINAL CORD, TOGETHER WITH ALL ITS PROTOPLASMIC PROCESSES AND THEIR DIVISIONS.

The axis-cylinder process with its divisions, side fibrils or collaterals, and the end ramifications (telodendrions, or motor end-plates) in the muscle represent parts of a single cell or neurone. a. h. Axone hillock devoid of Nissl bodies, and showing a tendency to fibrillation; a. x. axis cylinder or axone, also indistinctly fibrillated. This process, at a short distance from the cell body, becomes surrounded by a myelin sheath, m, and a cellular sheath, the neurilemma, the latter not being an integral part of the neurone; c, cystoplasm showing the dark colored Nissl bodies, separated from one another by the lighter ground substance; d, protoplasmic processes (dendrites), containing Nissl bodies; n, nucleus; n', nucleolus; n.R., nodes of Ranvier; s.f., side fibril; n of n, nucleus of neurilemma; tel., motor end-plate or telodendrion; M', stripped muscle fibre; s.L., segmentation of Schmidt-Lantermann. (From "The Nervous System and Its Constituent Neurones," by Lewellys F. Barker. Courtesy of Wm. Wood & Co.)

pedicles of the vertebræ. This combination of a sensory incoming (or centripetal), and an outgoing (or centrifugal) motor nerve is the type-form of all the spinal, and those of the cranial nerves that are called mixed nerves, viz., the 5th, 9th, 10th, and 11th.

These motor fibres now go to such parts as they furnish with motor impulses, and end in tendon, and muscle, and furnish the motor fibres of the sympathetic nerves to the viscera, and vessels of all kinds, terminating there in the end-plates peculiar to the locality and the structure. Nerve-fibres never unite with others, nor terminate in another, but run from the motor-cell in the anterior horn to some form of separate distribution in the structure which they are to energize. As the fibre approaches its destination it divides dichotomously (by twos), always at a node of Ranvier, and finally loses both its neurilemma and myelin sheaths. The axon or axis-cylinder then divides into a number of branches which become directly and intimately associated with the tissue-cells. The terminations vary with the situation and are termed as variously, end-organs, terminal organs, or end-tufts.

In skeletal muscles the axon penetrates the sarcolemma, and then divides into a number of small branches, which appear to be imbedded in a relatively large mass of sarco-plasm and nuclei, the whole forming the so-called "motor-plate." Each muscle fibre possesses one such plate.

In visceral muscle the nerve-fibres derived from sympathetic or peripheral neurons are primarily non-medullated. The axons divide and subdivide, and form plexuses which surround the muscle-cell bundles. Fine fibres are given off, which ultimately come into relation with each individual cell, on the surface of which they terminate in the form of one or more granular masses.

In the glands, the mammary for example, these same sympathetic or peripheral nerve-fibres pass into the body of the gland, and ultimately reach the acini, on the outer surface of which they ramify, and form a plexus. From this plexus fine fibres penetrate the acinus-wall, and end on the gland-cell. The fibres present a varicose appearance. (Brubaker).

In tendons it ends in numerous leaf-like expansions. The sensory-nerves gather impressions from their end-organs, or inter-epithelial arborizations.

On review it will be seen that motor nerves go directly from the cortex to the motor-cells in the anterior horn, and the new axons from thence directly to the end-plate or terminal in the structure which it is designed to innervate, while the sensory nerves terminate in the posterior horn, or in some nucleus in the cord or medulla, and that the new axon is soon interrupted by some nucleus. This indicates that a spinal motor tract is a device to convey impulses to the motor cells of the anterior horns from the cortex, which is equivalent to saying that this impulse is sent to the anterior horn cells in response to the will. The motor cells of the anterior horns stand always ready to obey this class of impulse, and also any other stimulus that may be brought to them.

We have seen that at every level some of the fibres of the posterior roots arborize about cells in the posterior horn, from which new axons go up to, and arborize about, the motor-cells of the anterior horn. They are just as competent to release the pent-up energies of these motor-cells as are the stimuli borne to them from the brain by the fibres of the crossed pyramidal tracts, so we see that an action may result from a desire of the individual (volitional stimulation), from pathological states of the cortical cells (involuntary stimulation), or from a peripheral irritation brought in by the sensory nerve (reflex stimulation).

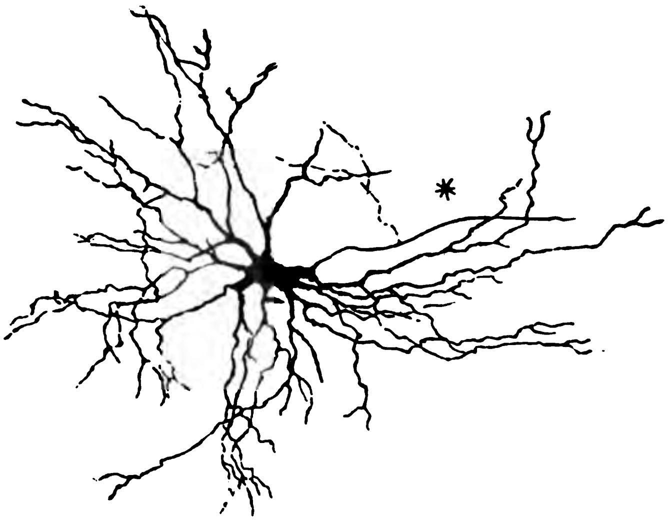


FIGURE 26. MOTOR OR GANGLION CELL.

From the anterior horn of the spinal cord of a human fœtus, 30 centimetres in length. A collateral branch (Golgi), from the axis-cylinder or neuraxon is seen; indicated by the star. Highly magnified. (Lenhossek.)

Every motion performed by the body falls within one or the other of these categories. The cerebellum is nothing more than an especially elaborate nucleus where impressions are re-arranged, and automatically modified, without the intervention of conscious thought. We see therefore that a movement may result from a desire to perform it—volitional. It may be systematized by the cerebellum after an initial impulse from the brain.

Such motor responses are called automatic, and are the result of some education of the structures. Dancing, or piano-playing are good examples of this sort of reflex action. The last class is where a peripheral stimulation from a part is carried by the most direct course to the motor-cells for that part, and evolves the desired motor response. This is purely reflex action, and accounts for some of the so-called reflexes which we use in the detection of disease of the nervous system. We are not far wrong if we consider all motion to be a reflex answer to peripheral stimulation, and brought about by the mechanism of the reflex arc, the simplest one being the sensory nerve to the cell of the posterior horn, thence to the motor-cell of the anterior horn, and thence to the motor end-plate by way of the motor part of a mixed nerve. The next one is where we take in the cerebellum, and the most complicated when the cortex of the brain is involved, but it is just as truly a reflex arc as in the first instance.

CHAPTER II.

GENERAL SYMPTOMS OF DISEASES OF THE NERVOUS SYSTEM.

If the nervous system is diseased, there will be disturbances of motion and sensation, and in the various phenomena arising from the imperfection of reflex action. Such losses will result in paralysis, or changes in the reflexes, disturbance of the control of the sphincters of the bladder and the rectum, or alterations in gait or posture, or defective sensory appreciation of peripheral stimuli, or ataxia, or pain, or trophic disturbances. Generally there will be some combination of these losses, but some of them may exist alone. Paralysis is the complete or partial loss of power in a muscle or group of muscles. If it involves a whole limb, or a part of it, the loss is called a monoplegia. If it involves a vertical half of the body, it is called a hemiplegia, but when all four limbs are involved it is termed a diplegia, carrying the implication that it is a double hemiplegia. This is also sometimes spoken of as a quadriplegia. These forms are generally the result of a cerebral or peripheral lesion. If the lower limbs are paralyzed, we say that the person has a paraplegia (of the parts below). Hemiplegia usually indicates that the lesion is in the brain, while paraplegia is the usual result of a lesion in the cord. Pott's disease, transverse myelitis, softening from thrombosis, hemorrhage or tumors of the cord, generally produce a paraplegia, but, if affecting the cervical region, will involve the arms also, and produce a quadriplegia. Primary lateral sclerosis, and syphilitic paraplegia are confined to the lower limbs. Minor grades of paralysis are termed paresis.

The differential diagnosis between paralytic conditions is much simplified if we class them into upper and lower neuron palsies. The upper (or cortico-spinal) neuron paralysis is due to a lesion of the cells in the cortex or their fibres, anywhere in their course from the cortical cell down through the brain, mid-brain, pons, medulla and cord, until they terminate in brushes about the dendrites of the motor-cell in the anterior horn at some level in the cord, and the same is true of a lesion of a cortical fibre to a motor cranial nerve if it occurs before its arborization about the cells of its nucleus in the crus, pons or medulla.

The lower (or spino-muscular) neuron paralysis arises from a lesion of the cell in the anterior horn of the cord, or of the motor-fibre at some point in its course from that cell to its termination in the motorial end-plate in the tissue which it innervates. No such clear distinction is possible in the case of the sensory neurons, since there is no such unbroken pathway from the peripheral tissues to the cortex. The broad distinction between palsies of the upper and the lower neurons lies in the fact that those of the first class deal with the volitional and inhibitory, and those of the second with the reflex pathways.

The upper neuron palsies have the following characteristics: the muscles are not completely paralyzed; all the muscles of a limb are affected to about the same extent; the limb is stiff (spastic); the muscles are rigid from unrestrained tonic contraction; they are oversensitive to a slight tap (myotatic irritability); tendon reflexes are increased; sensory reflexes are often exaggerated; atrophy is absent, except such as would be expected to arise from the disuse of a limb (disuse atrophy); the muscles are not flabby, since nutrition is not much impaired; the circulation is somewhat deficient, and therefore the limb is somewhat cold and blue, and there may be some edema of the extremity; sensory disturbances are absent, or slight, since, if present, they arise from associated damage to the sensory tracts; there is no reaction of degeneration to electrical currents.

The lower neuron type paralyzes the muscles fully at the outset; recovery is often impossible, or slow and imperfect; the complete type of paralysis is soon followed by a selective form in which single muscles, or groups of muscles, are the sole seat of the paralysis; the limb is never stiff, but is limp and sways by gravity (flaccid); tendons and muscles are so relaxed that the joint-surfaces become separated; myotatic irritability is absent; the tendon reflexes are lost; atrophy is complete and rapid; there is the reaction of degeneration; the circulation is always impaired, as the vessels are relaxed, the blood-pressure is reduced, and the capillary circulation is slowed; the limb is colder than in the first form, it is blue, and, while it sweats at first, remains cold to the end; sensory disturbances may not be present. This form is found in anterior poliomyelitis, both acute and chronic, amyotrophic lateral sclerosis, myelitis, either localized, or extending through the cord, syringomyelia when it invades the anterior horns, tumors and hemorrhages within the cord, softening due to thrombosis and embolism, and is also present in neuritis. The diagnosis of the site of the lesion whether it be a neuritis, or a damage to the cord, rests upon the distribution of the resulting paralysis, and the sensory losses. Muscle-groups, and not special muscles, are represented in segments of the cord, while definite muscles are innervated by nerves whose distribution is well known. The anesthesia from a segmental lesion is of a part or section of a part, while from a nerve lesion it involves definite small areas. Lesions like amyotrophic lateral sclerosis may involve both the upper and lower neurons, and then the type of paralysis is not sufficient for a diagnosis. We must here investigate the order of the appearance of the symptoms, since at some stage of the disease it will be purely of one or the other type. General inflammations of the cord will always disturb reflex action, and the action of the bladder and rectum, and produce marked atrophies. While therefore some parts of the body may show the symptoms of one type, and another part those of the other, the disease will finally involve all parts in a paralysis of the second, or lower neuron, type.

Injuries to the spinal cord by fractures, or injuries of the vertebræ, may crush the cord, or completely sever it. There is a diagnostic difference in the resulting conditions.

If the cord is partially severed, there will be total paralysis, which may be asymmetrical; below the site of the injury, rigidity of limbs; increase in the reflexes; disturbance of the sphincters, either causing retention, or spontaneous evacuation; probably some tympanites.

If the cord has been completely severed, there will be total paralysis below the point of injury; the limbs will be relaxed, and not rigid; the reflexes will be abolished; the paralysis will be symmetrical on both sides; retention of urine always; tympanites; vaso-constrictor paralysis, with the result that there is a dilatation of the subcutaneous veins, leading to an increase of the temperature of the part, and to priapism; increase in the genital reflex (tenderness of the testicles to pressure); loss of sensibility to pain and temperature, and usually to touch also, at levels corresponding to the segments injured. In the absence of these symptoms, we may conclude that the cord is not entirely severed.

LOCALIZING OR FOCAL SYMPTOMS OF DISEASES OF THE BRAIN.

Lesions of the brain produce certain symptoms by which we are able to fix the site of the damage, in addition to the general symptoms before stated, which denoted that the brain had been injured, but gave no clue to the particular structures which had been compromised.

These localizing symptoms are both motor and sensory, of which the former are by far the most valuable, since, while there is a pathway from the periphery to the sensory cells of the cortex, it is by no means the simple linkage which we encounter in dissecting the route of motor impulses. Sensory impressions meet so many cell aggregates in their upward course, in each one of which there is a possible modification in direction or character of impulse, that the final destination, and certain route, are as yet somewhat matters of conjecture.

The possibility of localizing a motor or sensory lesion in the brain depends upon the following facts:

1. That the brain is that part of the nervous system in which we originate voluntary movements. Therefore if a member of the body is capable of motion from some peripheral stimulus, but refuses to move in response to the will, we know that the neuron whose cell is in the brain is injured in some portion, and some added data will often definitely localize the site of the injury. Since in a definite area of the cortex is localized the cell-area capable of inducing every known movement of which the body is capable (for the cortical areas represent movements, like grasping for instance, and not separate muscles), the paralysis of a single member, or part of it, shows us, if the reflexes are exaggerated (the mark of a lesion of some part of the upper motor neuron), that the lesion is cortical. Monoplegia, with increased reflexes, is therefore the mark of cortical damage. Since the fibres from the widely distributed cells of the cortex are gathered fan-wise, to appear as a small bundle in the internal capsule, the amount of one side of the body which will be paralyzed by a lesion of a given size increases as we pass from the cortex to the capsule. When the capsule is reached, we find, therefore, that the type is hemiplegic, instead of monoplegic.

2. The intellectual processes are the result of the comparison and correlation of impulses from many parts of the brain, and when the process is completed, the act results from the stimulation of the area in which is stored up the muscular memory of the desired act. These are localized in different areas, and if the tracts leading to those areas are cut off the motion will not ensue, since the appropriate centers cannot coördinately be stimulated.

Speech has been supposed to be an instance of such groupings of specialized centers, but under the influence of Marie, who considers the actual loss as one of association from intellectual defects, these ideas have been modified.

3. The motor tracts in their downward course are brought into close relations with other structures, and the vertical site of a lesion below the internal capsule causing a motor loss can be ascertained by a consideration of the associated losses.

Sensory Lesions. The cells for the appreciation of general sensation lie mostly behind those of motion, but are not absolutely isolated from them. The motor-area has some sensory cells, but the proportion decreases as we approach the parietal cortex, which is purely sensory. It may be said with a fair amount of definiteness that the cells for sense-perception are located in the middle third of the posterior central convolution, and the inferior parietal lobe, and that the face-area is in the lowest, and that for the leg is nearest the vertex. (Typical lesions of the parietal lobe are hyperesthesia, impairment of muscular sense, ataxia, and asteriognosis. C. K. Mills, Journal of Nervous and Mental Diseases, July, 1909.) The theory of Ferrier and Mills locates them in the hippocampal region, but this is from experiments on animals, and it does not apply to man. This localization applies to all varieties of sensation, touch, muscle-sense, heat, cold, and pain. These several impressions are received by separate cells, so that one set appreciates touch, another the position of the limbs (this seems rather generally distributed in the central lobes), another pain, another heat, and still another cold, but steriognosis (the appreciation of the contour, weight, shape, and peculiarities of surface of an object) requires the perfection of all of these. When, therefore, any one of these fails, the individual will suffer to some degree from asteriognosis. The fact that there is a sensory loss will not in itself be localizing, but since, as I have said, parts of the body are localized on the cortex, we may assume that the paresthesia of a single part is the result of a cortical lesion. The sensory fibres are gathered into a bundle, which passes in the posterior third of the posterior limb of the internal capsule, therefore as the motor loss from a cortical lesion was a monoplegia, so a similar sensory loss is the paresthesia of a single part, and as the motor loss from a lesion of the capsule, or tracts

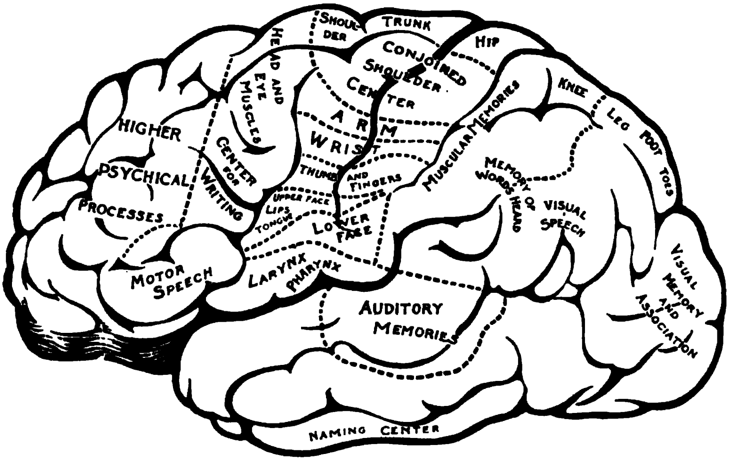


FIGURE 27. LOCALIZATION ON THE CORTEX OF THE BRAIN.

within the brain, was a hemiplegia, so from a sensory lesion in a similar location we may get a hemi-paresthesia, but not so certainly, since sensory representation is much more bilateral. As has before been said, destruction of areas or tracts produces paralysis of motion or sensation, while irritation of them produces exaltation of function, either muscular spasm, or pain in some degree; it may be severe pain in the other half of the body. The usual result of injury to the sensory cells is a paresthesia rather than an anesthesia, in the form of a slight numbness or tingling in the extremities, hands, and feet, and lips. Whether it be a decrease or an increase, it fades away into areas of normal sensibility. This differs from hysteria in just this indefiniteness of boundary, and also that there is no accompanying paralysis, and in hysteria it is often associated with a concentric diminution of the visual fields. While the loss is possibly for all varieties of sense-perception, it is often a dissociated one, and mostly of touch, and appreciation of the position of the limbs. The paresthesia from a cortical lesion is never absolute, and while it may arise from the organic damage due to a hemorrhage or the pressure of a tumor, it may also be functional, even if there is damage of some sort in the vicinity. It is a frequent warning of an attack of Jacksonian epilepsy, or of idiopathic epilepsy, and often is circulatory from temporary arterial spasm, as well as from the more serious disturbance which culminates in hemorrhage or thrombosis.

SUBCORTICAL LESIONS. If we could fancy ourselves looking at the under surface of the cortex of the brain, we should see a maze of nerve-fibres, tending downward from all parts of the hemisphere, to finally appear as a narrow band between the lenticular nucleus on the outside, and the optic thalamus to the inside posteriorly, and the head of the caudate nucleus to the inside anteriorly. This band is the internal capsule, and these are the neuraxons on their way to their distribution to all parts of the body. These fibres rotate somewhat in their course, and appear in the capsule arranged as pictured in Figure 24. Passing still downward, the motor fibres appear as a yet narrower band in the foot of the crus, and thence they pass into the pons. Here they are spread out somewhat widely upon the anterior aspect, passing through a sort of reticulum made by the interlacing of the fibres of the two middle peduncles of the cerebellum. Proceeding from the pons, they are again gathered into a bundle called the anterior pyramid, on the anterior aspect of the medulla. At the lower border of the medulla, the anterior pyramid of the left side crosses to the right, and the right pyramid crosses to the left in a similar manner, forming what is known as the motor decussation. After this crossing, the motor tract assumes a position just anterior to the posterior horn in the lateral portion of the cord, and is now known as the lateral tract, or the crossed pyramidal tract. It is called the crossed pyramidal tract in contra-distinction to about one-tenth of the original anterior pyramid of the medulla, which in most cases does not cross, but continues downward in the anterior part of the cord on the same side of the center line which it formerly occupied in the medulla (column of Turck). After leaving the capsule, the tract is always in close proximity to cranial nerves or their nuclei, or to the cerebellum, and therefore a lesion of the tracts might reasonably be expected to damage some other structure, and vice versa. The accessory damage therefore furnishes the grounds upon which we localize vertically a lesion of the motor tract below the internal capsule. Before passing to these lower lesions it should be pointed out that the fibres are so concentrated in the capsule that a small lesion will produce a damage of much greater extent than one of the cortex, or one between the cortex and the capsule, while the fibres are still more or less widely fanned out in that area, where they are collectively termed the corona radiata.

In the capsule a lesion must be exceedingly minute to produce a monoplegia, and such a result would be highly improbable, since a lesion the size of a pea might produce at least a temporary hemiplegia. A monoplegia might possibly result from a very small lesion in the crus, or the upper part of the pons, since the arteries are very minute, but since the fibres are more widely spread out in the lower part of the structure, a lesion of the same size in the lower part of the pons would result in a paralysis of more limited area. Lesions of the central ganglia are more sensory than motor, and resulting symptoms of both varieties will be discovered at the same time. The site of a lesion in the capsule will be shown by the portion of the body which is the seat of actual paralysis when the temporary general paralysis has passed away. The sensory fibres pass through the posterior portion of the posterior limb of the internal capsule, as is shown in Figure 24. In this area the fibres of special sense are massed in bundles which lie toward the anterior portion of the sensory area. These bundles are to some extent from both hemispheres, and a lesion must be in the capsule or the cortex to produce any permanent losses. If the sensory fibres are injured in the internal capsule the loss or change applies equally to all forms of sense-perception, which aids us in localizing the lesion, since lesions in the cortex, pons, or medulla, cause dissociated losses, i. e., one or more varieties of sense-perception, while another will remain normally acute. A finer localization applying to any level is sometimes possible in the case of slowly developing lesions. If motion is paralyzed, and, later, sensation also, we know that the common cause was first in the motor area, and had extended in the direction of the sensory tracts, or the opposite condition might be the fact. Hemianesthesia is generally due to a lesion in the posterior part of the internal capsule, or where the fibres are mixed with the motor ones, and is usually attended by hemianalgesia, or hemithermo-anesthesia. It is rarely absolute, on account of bilateral representation. As a rule the entire opposite half of the body is anesthetic to some degree after a sensory lesion in the capsule. By as much as a lesion lies above the capsule, by so much is it less competent to produce a hemianesthesia, and in the centrum semiovale there is apt to result an anesthesia of one limb, or part of the body also. The same losses, exaltations and perversions of sense-perception may be produced by lesions of the sensory tracts, although there is no single, long, uninterrupted tract like that of the motor system. We may find from any such lesion an anesthesia, or peculiar hyperesthesia, or both may be so combined that with an anesthesia the result of stimulation is a disagreeable sensation which may rise to a degree of pain if the stimulant is strong enough. The skin and mucous membranes anywhere may be the seat of this perversion, although the cornea is rarely affected. There are perfect sensory reflexes in the cord and ganglia, all the way from the lowest portion of the cord to the cortex. If this sensory perversion is present, there will therefore be reflex symptoms of irritation shown in cries and reflex movements, designed to remove the part from the irritant which would necessarily be present if such sensory impressions were received when the organism was in a state of health. The respiration and pulse may be quickened, and the face may show distress. This is assumed to be reflex, since all the phenomena will be present, if the brain has been previously removed. It is not necessary that the sensory cells should be injured. An injury to the tracts will exalt or depress these sense-perceptions according to the degree of injury.

CRUS. The peculiarity which localizes lesions in the crus arises from the fact that the 3d nerve (motor oculi) comes out from the inner side of the crus, and the optic tract lies upon it. A lesion in this locality which is extensive enough to cause a hemiplegia will in all probability involve the 3d nerve, and also affect injuriously the optic tract. We should therefore expect with the hemiplegia a paralysis of the motor muscle of the eye (except the superior oblique, and the external rectus), and probably a hemianopsia. A lesion at this point gives rise to the syndrome of Benedickt and Weber. In this the eye on the side of the lesion is affected, and therefore the oculo-motor palsy is opposite to the paralysis of the face and body. The eye is turned outward, and cannot be turned upward, downward, or inward, the pupil is dilated, and there is ptosis, since the lid falls from the paralysis of the levator palpebræ superioris. The corpora quadrigemina are so near that they can hardly fail to be affected, and the red nucleus within the crus as well. If the posterior corpora quadrigemina are involved there is disturbance of hearing and of coördination, and the latter result will follow a lesion of the red nucleus. The sensory tracts, tegmentum, and fillet, lie just above the motor fibres in the crus, and they are generally involved. If they are injured, there will be an anesthesia of the opposite side of the body without any disturbance of the special senses, but if the acoustic fibres in the tegmentum are injured, there will be deafness if the lesion is bilateral. In lesions below the internal capsule there may be a loss of the perception of pain or temperature, accompanied by paralyses of some of the cranial nerves, whose known point of origin localizes the injury causing the loss of these perceptions.

PONS. A small lesion in the upper part of the pons may be indistinguishable from one in the internal capsule, lesions in the lower part of the pons, only, being characteristic. A combination of paralysis of the limbs and body, with palsy of some of the cranial nerves is diagnostic of lesions in the pons and medulla. The lesion here is generally a hemiplegia, since the raphe is strong enough to confine an ordinary hemorrhage to one side of the structure. If this is ruptured, a diplegia will be the result. Localization of a lesion in this locality is from the coincident paralysis of some of the cranial nerves. The 5th, 6th, and 7th cranial nerves run in this structure from the time that they leave their nuclei of origin, until they emerge upon its surface. The nuclei lie in the upper part of the pons. They are very liable to injury in any lesion of the pons. Crossed or alternate hemiplegia is especially a sign of a lesion in the lower third of this division of the brain-stem. This means that there is a paralysis of the face upon the same side as the lesion, and of the body upon the opposite side. The fibres from the brain to the facial nucleus lie upon the side of their origin in the upper part of the pons, but cross in the pons to gain the nucleus on the opposite side in its lower part, and therefore a lesion in the upper part results in an ordinary hemiplegia. If the lesion is in the lower part, the nucleus of the facial and its root-fibres are involved on the same side as their distribution, and therefore an alternate hemiplegia results. The nucleus of the 6th nerve (external rectus) is practically always involved, and therefore the eyes will turn away from the lesion, which is contrary to the rule when the lesion is higher up. If the *nucleus* of the facial is damaged, *the eye cannot be closed,* but if *the fibres* are injured on their way to the nucleus it *can be closed.* The pupils are contracted to a pin point. The tegmentum (or dorsal portion of the pons, composed of sensory tracts, and of cell-groups) lies above or behind the motor tracts, and if the lesion is extensive enough to involve it, there will be anesthesia in the area of distribution of the motor palsy. The motor and sensory portions of the 5th nerve lie here, and may be involved separately, or in combination. If the sensory fibres are alone affected, we shall find anesthesia of the face on the side of the lesion, and a paralysis of the limbs on the other, from an implication of the motor tracts, but if the whole nerve is involved there will be the same sensory loss as above, and a crossed motor paralysis. Spasms and convulsions occur when the pons is affected, even in acute diseases. The middle peduncles of the cerebellum lie on the under side of the pons, and many pontile symptoms arise from a simultaneous involvement of this structure. If the middle cerebellar peduncle is invaded there are vertigo, and vomiting, and tinnitus is usually present. Deafness may occur, and if the lesion is irritative, and not destructive, there may be gyratory movements, and forced one-sided positions of the limbs in lying, which may be accompanied by flexion of the head and eyes to the same side, but not necessarily to the same side as the lesion.

MEDULLA. Lesions here compromise some one or all of the nuclei of the 9th, 10th, 11th, and 12th cranial nerves. If the lesion is unilateral, we shall find a crossed hemiplegia, so far as the function of these nerves is concerned, and dysarthria with hemiplegia has been known to occur. The cerebellar tracts enter that organ through the restiform bodies, and these tracts are joined by fibres from the nucleus cuneatus and gracilis, and also from the olivary bodies. If, therefore, there is a lesion of the lower part of the medulla, it will cut off the tracts to the cerebellum, and give rise to vertigo and loss of equilibrium, but if above these tracts no such losses will exist. Clinically lesions of this area are so rapidly fatal that the theoretical results are not often seen. Owing to the close relation of the nuclei of both sides in the medulla, a small lesion usually produces bilateral symptoms. A characteristic lesion of the medulla is a labio-glossal-laryngeal paralysis, due to a very slowly progressive bilateral degeneration of the motor bulbar nuclei. It usually starts in the nuclei of the hypoglossal, next affects the spinal accessory, next the glosso-pharyngeal, next the pneumogastric, and occasionally the facial.

BASAL GANGLIA. It has seemed best to treat such lesions separately from others which are also subcortical, since all statements of a localizing significance must be made with many reservations. Most of the symptoms occurring in case of their injury or disease must be ascribed to a simultaneous injury of the tracts in the vicinity. The following functions have been ascribed to these ganglia, but somewhat inferentially. The caudate nucleus is supposed to have some relation to the movements of the legs, while the lenticular nucleus is similarly related to the movements of the arms, the act of eating, and the function of speech. The optic thalamus is known to be a sensory ganglion, and it has been claimed that every sensory tract ended in the thalamus before its ultimate radiation to the cortex. It is certain that a lesion of the pulvinar will cause only hemianopsia (a one-sided loss of vision), and this explains why the destruction of one thalamus produces no lasting anesthesia, since sensation has a bilateral cortical representation. A lesion of this body has been assumed to be the cause of a hemi-chorea, or hemi-athetosis, but both are now believed to flow from an irritant lesion of the neighboring motor or sensory tracts. When the facial muscles have been paralyzed to voluntary impulses they will often be found to respond to the calls of the emotions, while on the other hand, if the thalamus has been injured, the muscles will respond to volition, but not to emotion. From this it has been concluded that the proper response to the emotions depends upon the integrity of the thalamus. The same exhibition of causeless laughter, excitement and depression results from a tumor in the frontal lobe, and not from such a growth in the corpus callosum. The similarity in result of a lesion in the frontal lobe, and the optic thalamus, is explained by the discovery that a bundle of fibres originating in the cells of the frontal cortex passes through the anterior portion of the internal capsule, and ends in the external nucleus of the thalamus. The person with a lesion in the thalamus cannot smell in the opposite nostril. Reflex thermic centers have been located in the basal ganglia, but not more exactly, as well as the vaso-motor, secretory, and trophic control of the opposite half of the body. Localization must be made by associated capsular symptoms.

Lesions of the external capsule and claustrum cannot be localized, but in some cases of paraphasia a lesion of those ganglia has been found in the left hemisphere.

CORPORA QUADRIGEMINA. These are visual and auditory paths, and the superior cerebellar peduncles contain the nuclei of the ocular nerves.

Lesions of the corpora quadrigemina alone are rare, as they usually are accessory involvements in lesions of the crus, or the anterior fossa. If the anterior pair were the site of a lesion, we should find oculo-motor palsy, loss of the pupillary reflex, and nystagmus. If the posterior pair were affected, hearing and coördination would suffer. A defective action of the oculo-motor nerve upon both sides is rare, and if it occurred it would point to a disease of the corpora; the same is true of the optic nerve, and if blindness is not a hemianopsia, nor due to optic neuritis, choked disc, or optic atrophy, it may be due to a lesion of the corpora quadrigemina. Both pairs are commonly affected in a discrete lesion, and a combination of characteristic symptoms would be diagnostic. Nothnagel says that there are only two focal symptoms which are absolutely diagnostic: 1st, a slowly increasing cerebellar ataxia, identical with that occasioned by that of the worm; and 2d, a gradually increasing, and not entirely symmetrical ophthalmoplegia, the superior and inferior recti being most often affected. Bruns says that the ophthalmoplegia most often precedes ataxia, by which it differs from a cerebellar lesion, where the ataxia is always first.

CEREBELLUM. Lesions of the cerebellum produce the following symptoms in the order of their frequency: Headache, vomiting, ataxia, asthenia, occipital headache, and tenderness of the neck with rigidity, an inclination to turn the body toward the side of the lesion, and convulsions. Because it sets up pressure on the pons and medulla, the latter particularly, there are some cranial nerve palsies from injury of the nuclei, polyuria, glycosuria, tremors and sudden death, these latter symptoms arising from pressure on the fourth ventricle. This organ preserves the equilibrium of the body, but has nothing to do with touch, pain, or temperature perceptions. The symptoms are practically those resulting from a reduction of the tonus to muscles when the lesion is destructive, shown by the asthenia, and by its irregular increase into spasm when there is irritation in the cerebellum. The faculty of coördination depends upon the perfection of function of the cerebellum, and when it is disturbed the coincident loss of tone and of coördination produces paresis, ataxia, asthenia, incoördination, tremors, astasia (motor incoördination in standing). Lesions of the lateral lobes have been found post mortem which have produced no symptoms during life, but if the central lobe or worm is the seat of a lesion very definite symptoms are sure to result. The functions of the organ seem to be initiated by it as a whole, but one side also seems to be able to assume the function of the other. The prime requisite for the production of symptoms from the lesion of one lateral lobe seems to be that it shall be sudden in its onset, and rapid in its growth like hemorrhage, abscess, or tumor of rapid developement. A sudden lesion of the lateral lobe results in weakness upon that side of the body, lack of coördination, and the patient is apt to stagger toward that side.

That a patient does always stagger to the same side is of no great clinical value, since it is impossible to state whether the lesion is irritative or destructive. It is also found that movements cannot be reproduced with rapidity and precision, and the member soon tires. This is adiadochokinesis (Babinski), and while indicative of cerebellar disease, is not indubitably so, but in a one-sided cerebellar lesion it is usually easily demonstrated. Catalepsie cérébelleuse of Babinski is suggestive of cerebellar disease. Flex the thighs on the body, the legs on the thighs, and separate the feet. After a preliminary tremulousness the legs will remain quietly in this position for a remarkable length of time.

The reflexes are reduced, and the patient tends to bend to that side. Weakness of the ocular muscles upon the same side is found, and nystagmus results from an attempt to turn them to the injured side. Lesions of the head of the worm produce a tendency to fall forward, and of the back portion a tendency to fall backward. The staggering and ocular disturbances are accompanied by a vertigo which is increased on assuming an upright position. A tumor of the frontal region induces a similar vertigo, and an occipital headache, and in one-fifth of the cases a similar bilateral weakness of the reflexes. Irritative lesions are marked by an exaggeration of these same influences over the musculature of the body. There is muscular stiffness of the side of the body affected, and there is nystagmus in which the jerking is toward the side of the lesion, and the body is strongly arched to this side. When the middle lobe is affected in the same irritative manner, or both lobes are simultaneously affected, there is opisthotonos, or emprosthotonos. If we have extension of a growth primarily in the cerebellum, but which later impinges upon other structures, the symptoms will relate to those structures secondarily affected. If on the motor tracts, we shall find spastic symptoms on the side opposite the lesion with increase of myotatic irritability, and a tendency to contractures. If it is on the floor of the fourth ventricle, the nuclei of the cranial nerves will be affected, and we may find paralyses of the fifth, sixth, seventh, eighth, ninth, tenth, and twelfth nerves. The eighth is particularly apt to be affected, and we may get symptoms like those of Ménière's disease. Labyrinthine disease may closely simulate a cerebellar lesion, and septic processes in the ear may extend to the cerebellum. Irritation of the fourth ventricle may produce polyuria, and glycosuria. Obstruction of the veins of Galen may set up a hydrocephalus, and sudden death results from pressure upon the nucleus of the pneumogastric.

DISTURBANCES OF THE SPECIAL SENSES:

VISION. The optic nerve is part of the brain, and diseases of the brain may produce blindness, hemianopsia (blindness of one-half of one, or of both eyes, and is homonymus if of both left or right halves, while it is heteronymus if it affects the left half of one, and the right half of the other eye), optic neuritis, choked disc, optic atrophy, or hemorrhages. Papillitis, or optic neuritis is an infiltration and consequent swelling of the head of the optic nerve, so that the clear outline of the nerve-head as it appears in the retina is clouded or blotted out. The nerve-fibres are normally so transparent that they pass out of the nerve and over on to the retina, without cutting off the outline of the circular aperture in the choroid through which the nerve passes. When they are infiltrated, they become translucent, and later opaque, and hide this margin, and the ophthalmoscopic image reveals a chrysanthemum-like body, choked disc, placed in this apertrue (see pg. 180). A steep prominence is thus formed, about two-thirds of a millimeter in height (refractive difference of two diopters), which is reddish, or reddish gray in color, and the arteries are contracted, and the veins are dilated. If the process is a severe one it leads to optic atrophy. From the cells of the cortex at the cuneus, the visual fibres are gathered into the bundle known as the radiation of Gratiolet, and pass forward into the posterior portion of the internal capsule, thence into the pulvinar of the optic thalamus, thence into the external geniculate body, thence into the anterior corpus quadrigeminus, and decussating partially in the chiasm are distributed to the retina. A lesion of the cuneus produces blindness of a sector of a circle. This is a rather theoretical result, since clinically we usually find a hemianopsia if there is a lesion anywhere in the occipital lobe, and it may produce a simple dimness of vision. If the lesion is subcortical it is usually accompanied by symptoms of localizing value. If it is near the cortex in the left hemisphere we shall expect to find with it some form of word-blindness, or aphasia, since the cortical localization of visual speech lies very near here, in the angular gyrus. If it is in the internal capsule, or near the optic thalamus, there is usually hemianesthesia, or hemiplegia combined with it; if in the external geniculate body, the fibres going to the anterior corpus quadrigeminus are involved, and the movements of the pupil are compromised, and we get Wernicke's pupillary reflex, which can only occur when a lesion is in the optic tract, or basal ganglia, and does not occur in lesions of the cortex. This reflex is that if light is thrown into the pupil on the blind side there is no pupillary response. When a lesion is confined to the anterior corpora quadrigemina there is no loss of vision, but the movements of the eyes cannot be ensured, and hence single vision is lost, and the person suffers from diplopia. This causes vertigo, and a staggering gait like that resulting from cerebellar disease will result if the red nucleus, lying under the quadrigemina, is also affected by the lesion, inasmuch as the red nucleus is intimately connected with the cerebellum. The lesion of an optic tract upon one side will cause hemianopsia, and presents Wernicke's sign also. Lesions of the crus will also cause hemianopsia associated with motor paralysis, oculo-motor palsy and ptosis. The result of lesions of the chiasm is described in the chapter upon cranial-nerve lesions. None of the lesions just described are competent to produce blindness of one eye, i. e., both halves, or a concentric diminution of the field of vision, but such conditions arise only from diseases of the eye itself, or of one optic nerve, and are visible with the ophthalmoscope, or are due to hysteria, and to be diagnosed by the results of a general physical examination. While persons learn through all the senses, some acquire knowledge more easily through one sense than through another, and so all are divided into visuals and auditives, according to that selective route of acquirement, yet it is a fact that the greater proportion learn through the eye than the ear. When therefore the cuneus is injured, especially upon the left hemisphere, the person often loses the power to recognize formerly well-known objects by sight, while the sound of the name may identify them to him. This is psychical blindness, or apraxia.

If the cortex of the angular gyrus, located at the junction of the occipital and parietal lobes, is injured, we get a modification of psychical blindness, since at this point are stored up the memories of words seen. This renders the person unable to read, and is called word-blindness (alexia). It is rare that such a lesion can occur, and not affect the subcortical tracts which lie just beneath. If these are injured, hemianopsia will result, and it may be accompanied by hemianesthesia, and hemiataxia, and if the lesion projects into the tracts running forward from the center for visual word-memories, the person may also lose the power of writing (agraphia). Alexia is therefore usually complicated by agraphia, hemianesthesia, and hemiataxia. The lesions of the visual cortex are naturally different in grade, and may be irritant, as well as of the destructive nature just dwelt upon. If the cortex in this region is irritated, the central stimulation will awaken memory images whose appearance would be ascribed to the accustomed peripheral stimulation of the retina. This central irritation gives rise to visual hallucinations, and is the probable cause of the hallucinations which are common in the delirium from all forms of intoxication, whether it be caused by alcohol, the poisonous metals, or from infections. They are unilateral as a rule, and may precede attacks of Jacksonian epilepsy, and constitute the aura in these cases. They may be simple flashes of light or a play of colors, or may amount to definite pictures of some complexity.

Disturbances of Hearing. In the discussion of these disturbances the same difference will be shown between irritative and destructive lesions which were displayed in the visual areas. Hearing, as a special sense, is bilaterally represented, and therefore deafness will not result from a unilateral lesion of the first, and part of the second temporal lobes where this sense is cortically represented. If however there is irritation of the cortex in this locality, upon either side, the person will suffer from auditory hallucinations, and the irritant may be organic change in its early stage, or an intoxication of any form. This accounts for the noises and voices heard in delirium, and also for the auditory auræ in the various forms of epilepsy. If these lobes in the left hemisphere are destroyed, we get a peculiar symptom-complex, since here are stored up the memories of words heard, and of musical harmonies. When these are lost the subject can no longer recognize objects by their spoken names, and cannot understand conversation, and since we sound words to ourselves before speaking them, as if we must correct our conception by a central auditory test, the person soon becomes a mute. This is amnesic aphasia, and the person speaks a jargon without meaning. This loss is not always symmetrical, any more than are the losses of sense-perception, and therefore a person may lose the memory of spoken words, and still retain that for harmonies. The loss, when confined to the appreciation of spoken speech, is psychical deafness. Since reading and writing are subordinate to emissive speech, they are both liable to be impaired when there is an auditory loss. The amount of the loss is variable, and varies from the same lesion in different persons, according to the amount of their acquirements. The losses are in inverse order to their acquirement. A person who is word-deaf will fail to appreciate the meaning of sentences, while he may still appreciate single words. The names of objects acquired first will persist longest, but nouns will generally be lost while they can still use adjectives, verbs, and prepositions. After they have lost the use of nouns, they may still be able to make themselves understood by the use of round-about methods of description of their uses or properties. Occasionally all word-memories are lost. The area for the storage of word-memories is a little larger than that utilized for hearing alone. Deafness is rare from a subcortical lesion on account of the fact of double representation before referred to. The cochlear division of the auditory nerve alone conveys sound impressions, and the fibres lead to the lemniscus of the same or opposite side, and the posterior corpus quadrigeminus. A lesion of one lemniscus cannot produce deafness (double representation again), but when both are affected deafness will result, and this has occurred in pontile lesions.

Disturbances of Smell. Such disturbances are rarely seen except in organic lesions, and in mental disease. The cortical representation is supposed to be in the uncinate gyrus of the amygdaloid nucleus, which is located at the tip of the temporal lobe, and taste is also, by some, located here. Irritation of this area will cause hallucinations of taste and smell, and such an aura sometimes precedes epileptiform convulsions.

LOCALIZING SYMPTOMS OF LESIONS OF THE SPINAL CORD.

Paralysis is in itself a localizing symptom, since the segment from which any given muscle is innervated can easily be found in the charts for localization. If, therefore, a muscle shows paralysis, that fact alone indicates the presence of a lesion in the muscle, nerve, or a definite portion of the central nervous system. A little further study will give grounds for a decision as to which one of the three just named is the actual seat of the lesion.

Reflex Disturbances. Tendon-reflexes suffer variation in two directions, viz., the reflex may be exaggerated, or it may be diminished or lost, and the variety of change exhibited will give a basis for localization. If exaggeration exists it may be from a direct irritation of the cells of the anterior horn from a local cause, or it may be from a general neurasthenic condition, or from a lesion of nervous structure somewhere above the cells of the anterior horn. If from direct localized irritation, the exaltation will be confined to a single reflex, and there will be weakness, and some degree of atrophy of the parts innervated from the segment, or segments, essential to this reflex. The localization of the lesion is then a simple matter. If it arises from a general neurasthenic condition, the simultaneous involvement of all the reflexes, and the associated symptoms proclaim the character of the process. If it results from a lesion of the upper neuron, the parts affected will show a moderate degree of generalized paralysis, without marked atrophy, or conspicuous changes in temperature, and there will be some grade of rigidity in the limb, which will localize the lesion in some definite part of the tract, from the cells of the anterior horn up to and including the cells of the cortex. We gain our knowledge of its vertical position from the fact that all the reflexes below the lesion will be exaggerated, while those above will be normal.

The reflexes will be lost in coma, conditions of shock, and sometimes in the invasion period of febrile disorders. Their loss under these conditions has no localizing value. They will be lost when the limbs are rigid from fibro-tendinous changes in the muscles, and here have no localizing value. They will be lost in section, or complete degeneration of the sensory or motor fibres, or of both, of a peripheral nerve. The loss may be localized in the nerve from the fact, that if in the motor fibres alone, there will be motor paralysis in the muscle to which it is known to be distributed. If it is in the sensory fibres alone, there will be sensory losses in areas of skin known to be innervated from this particular nerve. The reflexes will be lost if the tracts in the posterior columns of the cord have been destroyed. The sensory losses in skin areas, discovered as before, with no correspond-

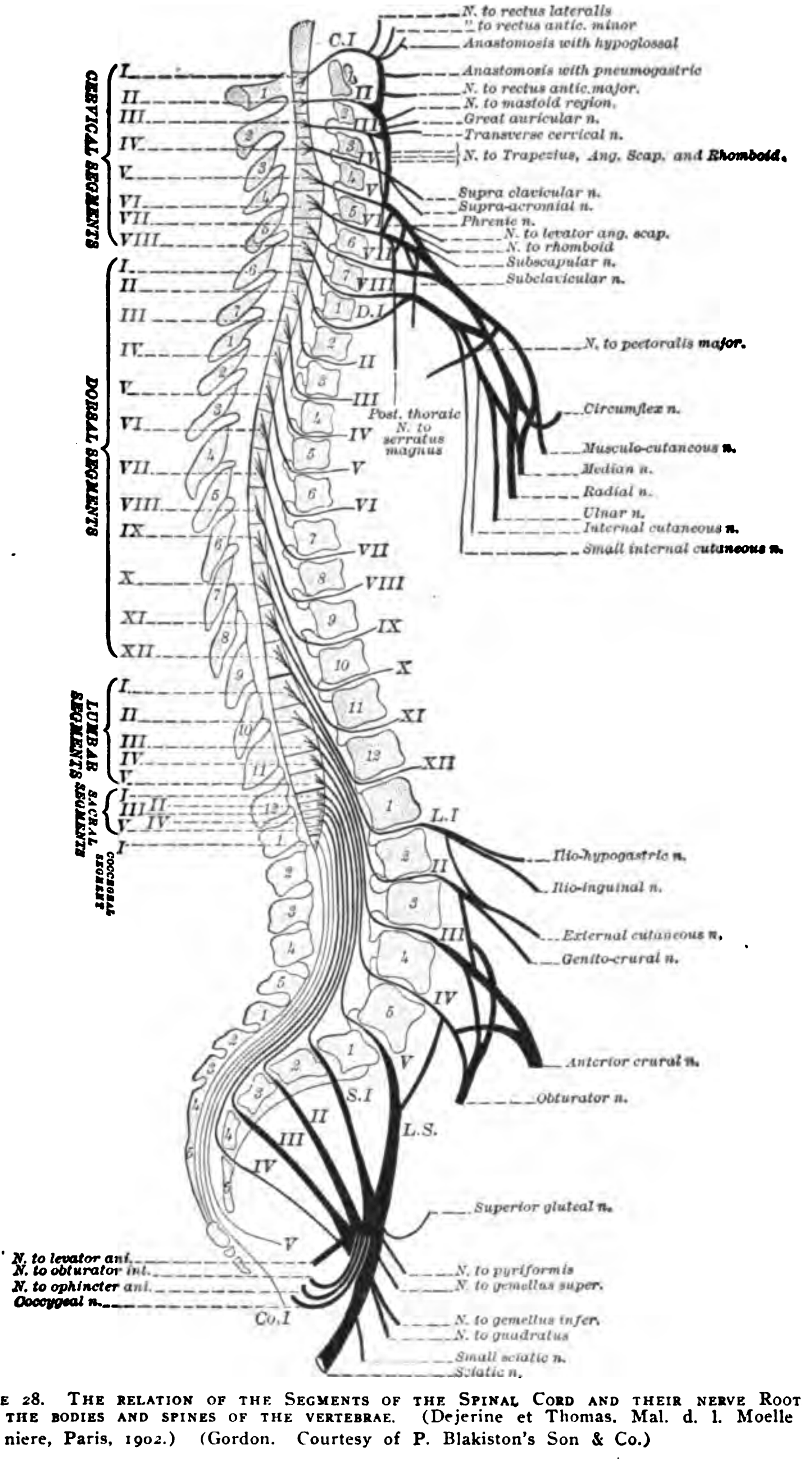


Figure 28. The relation of the Segments of the Spinal Cord and their nerve Roots to the bodies and spines of the vertebrae. (Dejerine et Thomas. Mal. d. l. Moelle Epiniere, Paris, 1902.) (Gordon. Courtesy of P. Blakiston's Son & Co.)

ing motor loss, will show that it is in the cord, since, being in a tract made up of fibres coming from many different levels, the sensory losses will be in patches in many separated parts of the skin in the region below the site of the lesion. Reflexes will also fail if there is destruction of the cells of the anterior horn in one or more of the segments making up the motor part of the reflex arc in question.

We can assure ourselves that it is not in the nerve, since the sensory function is not disturbed, and trophic symptoms are present, indicating a localization in the anterior horn, and we can select the definite segment from the fact that we have charts which point out the segment from which any given muscle is innervated, or from which a given reflex is initiated. Loss of the reflexes will also occur when there is a hemisection of the cord involving one lateral half. We know that it is of one half of the cord from the fact that the side on which is the lesion will be paralyzed, while the opposite side of the body will be anesthetic, but the reflexes will be lost on both. This is the Brown-Sequard syndrome. The vertical level of the lesion will be revealed from the fact that the next segment above the site of destruction will be irritated, with the result that there will be a zone of hyperesthesia in its area of innervation, and from the charts the segment may be identified. (See page 79.) If the cord be completely severed there will be complete loss of the reflexes below the lesion. The reason for this loss in place of expected exaggeration, as is the result of the release of inhibition in partial losses, is not certain, but has been ascribed to loss of tonus.

Skin-reflexes are not well enough understood to be of as great localizing value as are the tendon-reflexes. They probably depend upon the integrity of definite centers in the cord, but their position is not known. Since they are not always present in health, and may be inhibited by volition, their value is still further diminished. They are never exaggerated by organic disease, but may be in general conditions which produce a condition of nervous irritability in the whole nervous system. They are never lost in hysteria, while they may be in organic disease, and it is a point in differentiation when that disease is in question. If the tendon-reflexes are increased they are commonly diminished proportionately, and so furnish data for an opinion of the presence of organic changes, and in a unilateral lesion may give information concerning the side of the lesion. Babinski's toe-sign (over-extension of the great toe on stroking the sole of the foot) localizes a lesion in the upper neuron, but will not define its vertical level.

Clonus may arise from a pretty severe lesion of the upper motor neuron, or it may arise from irritation of the motor cells in the anterior horns of the cord. If it arises from an upper neuron lesion it has no further localizing significance, but if from a lesion of the horn-cells, the clonus will be limited to the sphere of influence of that segment. It is then localizing to a definite level, and it also excludes upper neuron disease.

MUSCULAR SPASM. This is possible whenever the upper neuron is affected, but does not follow disease of the anterior horn-cells, or of the muscles. If we find that the limb becomes rigid on attempts at motion, sometimes when passive, we may deduce that there is a lesion somewhere in the upper neuron. This spasm may be alternately of the flexors and extensors, causing the limb to shake violently. This has been called spinal epilepsy, although not at all epileptic in fact. Vertical localization is only possible when certain higher levels furnish symptoms for exclusion.

FIBRILLARY TWITCHINGS. Here the whole muscle does not contract, but a few fibres here, and others there, making a flickering on the surface of the muscle, but may be numerous enough to disturb its balance. It never arises from disease in the muscle, and an irritant lesion of the upper neuron throws the whole muscle into spasm, and we therefore know that the cells of the anterior horn are beginning to degenerate, and from the charts we can discover the segment which is affected.

CONTRACTURE. This has a localizing value, if it is of a single muscle, or part, but as a rule only as a factor in the history of the case.

ORGANIC REFLEXES OR BLADDER AND RECTAL CONTROL. Lesions of both the upper and lower neuron will disturb the control of the sphincters of the bladder and the rectum, and therefore the localizing value is small. If however the reflex center in the cord is destroyed the detrusor will be paralyzed, the bladder will be over-distended, and finally overcome the power of the atonic sphincter, and cause a constant dribbling of the urine. This is called passive incontinence, and is somewhat significant of such a lesion. It may be of value to remember, that while retention and suppression of urine are common in hysteria, incontinence is not a symptom.

SENSORY DISTURBANCES. Sensation may be disturbed by a lesion of the structures to which the sensory nerve is distributed, by a lesion of the peripheral nerve, by a lesion of the posterior roots, by disease of the tracts and cell-areas in the cord, and at any point in their further course up to and in the brain. After we pass above a definite level in the cord, the localization is only possible by the study of the entire symptomatology of the case. If the loss is due to a local lesion of the surface, the reason of the disturbance is apparent. If it is due to disease or injury of the peripheral nerve, it will be for all varieties of sense-perception, and there will be an accompanying paralysis, or if the branch is entirely sensory, the definite area of loss will furnish the requisite data for its localization. If the nerve-roots are involved the fact is indicated by the character of the results, and

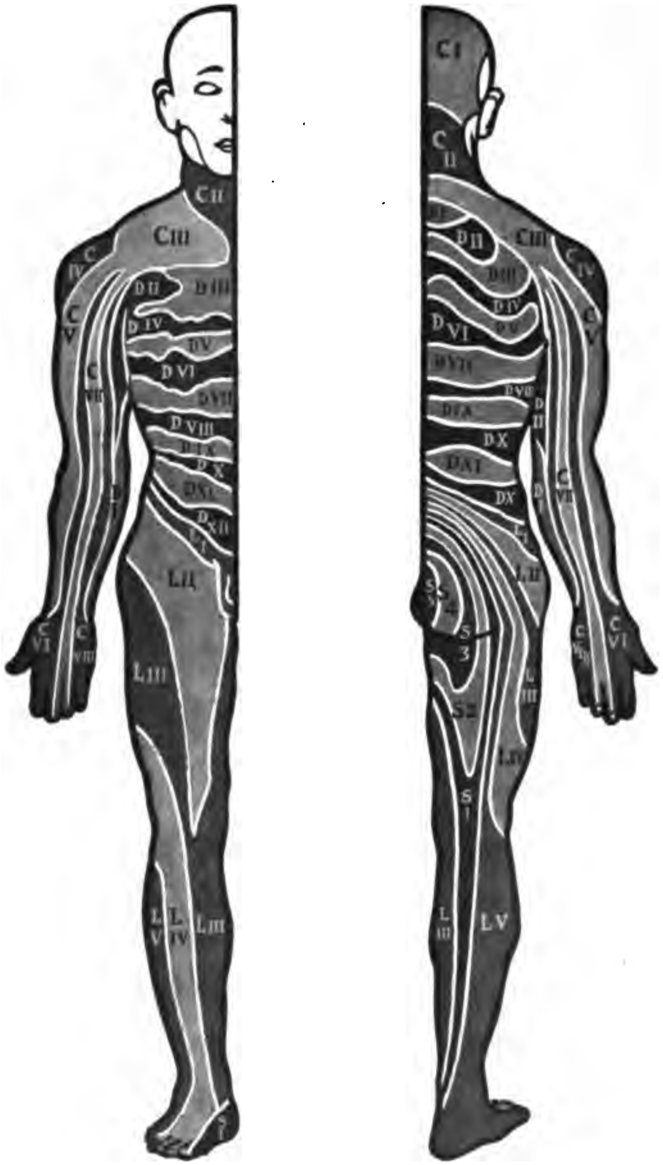


FIGURE 29. AREAS OF INNERVATION OF THE SPINAL NERVES.

The numerals, with the letters, refer to the spinal segments from which these areas derive their sensory innervation. (Modified from Starr.)

the vertical localization is indicated by the area of distribution of these symptoms. Conduction of sensory impressions from parts below is impaired in an increasing degree, and reflex action of the cord at that level is compromised. Pain is a prominent symptom, and is felt in any of the structures innervated from the given level. This pain may be felt in areas which have lost common sensation. Girdle-pains have their origin in this condition, as well as in more central lesions in the posterior columns, and are sensations of constriction in a more or less definite band about the body or a limb. If the fibres are affected immediately after their entrance into the cord, the result will be a loss of sense-perception in the parts below, and will generally be for all qualities, since at once the fibres for all sensation are gathered into a narrow band on the external surface of the columns of Burdach. This universality of loss will point to a localization of the lesion in the posterior columns, since the fibres almost immediately become widely separated in their further course to the tracts in which they ascend. Since sensation to any part is subserved by the associated action of at least three contiguous segments, localization must always include one segment above that designated as the one supplying any particular area. If we find that equilibrium or accuracy of motion (static ataxia, or incoordination) is exclusively affected we may localize the lesion in the direct cerebellar tracts (it is never there exclusively except from tumor), while if it is a loss solely of the perception of pain and temperature, we should fix the loss as possibly in the cells about the central canal, or in the anterolateral tracts which are the upward paths from these cells. In any case we can fix the upper level alone of the lesion. Pain in all these cases, except from root lesions, is localized in the areas of the distribution of the involved nerves, and never in the back, pain in which is caused by root lesions, or general neurasthenic conditions, or hysteria, and in the latter case is movable by suggestion.

LESIONS OF THE CAUDA EQUINA. These are like those of a mass of peripheral nerves, and so are segmental in distribution. The inner fibres alone suffer from the slighter grades of violence, and the outer also only when the injuries are more severe. To diagnose this lesion from disease in the segments the examination must reveal a physical disturbance of the vertebrae or sacrum else we must conclude that the symptoms arise from slow compression from some internal cause, as a tumor for instance. If merely the lowest roots are injured, the lesion will be restricted to the coccygeal nerve, and the only symptom will be a paralysis of the levator ani, with anesthesia of the anus and perineum, the sphincter of the anus being intact. If the lesion is below the level of the second sacral root, the anesthesia of the skin is diagnostic, being saddle-shaped, including in its upper part the skin about the anus, perineum, and posterior aspect of the scrotum and penis, with a small anesthetic area running downward from the perineum along the posterior internal aspect of both thighs. The mucous membrane of the urethra and bladder will also be anesthetic. The bladder and rectum are paralyzed, and the anal reflex is lost. In both these and the preceding lesions there is no muscular paralysis of the lower limbs, and the knee-jerk and plantar reflexes are normal. If the lesion is below the third lumbar vertebra, the third lumbar roots escape damage, the anterior crural and obturator nerves will escape, the sciatic, gluteal, and pudic nerves will be involved. The muscular paralysis and atrophy will affect only the muscles supplied by the last-named nerves, and therefore there will be paralysis with wasting of the glutei and hamstring muscles, and of all the muscles below the knees. The anus and bladder will be paralyzed and there will be anesthesia in the corresponding root-areas, but the knee-jerks will remain normal. If the lesion is just above the first lumbar root there is paralysis with wasting of all the muscles of the lower extremities, anesthesia of the lower extremities, buttocks, perineum, and genitals, with the exception of the inguinal regions, and the upper and anterior part of each thigh. There will be a paralysis of the bladder and rectum, with loss of sexual power, and loss of the knee- and ankle-jerks, and of the sphincter and anal reflexes. If the whole cauda equina is involved, the paralysis will be that just described, but the anesthesia of the lower extremities, buttocks, and genitals will be complete.

GAIT. The gait of a patient is often diagnostic so far as concerns its classification as paralytic, spastic, or ataxic. The paralytic gait is produced only by a lesion of the lower motor neuron, but mere inspection of the gait will not certainly reveal the closer localization of the lesion. This gait may result from a lesion in the anterior horn, or of the peripheral nerve, and will have the accessory symptom of rapid wasting of the muscles. In both these cases the whole limb must be dragged along, and if typical, the patient will be obliged to make use of crutches or a cane. If the lesion is in the cells of the anterior horn there will be a preliminary paralysis of the whole limb, which will be followed by a definite paralysis of a group, or of several groups, of muscles. All the muscles of the limb may be paralyzed permanently if the lesion in the cord is sufficiently widespread, but clinically the condition is rarely so extensive. In this case the articulations are relaxed, and the joint surfaces become separated, the feet turn in, and the big-toe is scraped along the ground. The marks, then, of horn-lesions are paralysis of the function of the limb as a whole, with flaccidity, marked atrophy, separation of joint surfaces, inversion of the foot, and scraping of the toe on the ground. If the peripheral nerve is affected, the limb as a whole may not be paralyzed, but the flaccid paralysis of a part may render it just as useless as an implement of progression. The common seat of such a lesion is in the anterior tibial, which results in "drop-foot." So far as the peroneal region is concerned, the phenomena are the same as just described. There is a flaccid paralysis with rapid atrophy of the parts involved, and there is relaxation of the joints. In this case, the fact that the peripheral part of the limbs is paralyzed, while the proximal part is competent, produces a characteristic gait. The upper part of the limb can be extended or flexed, while the lower part hangs loosely. In walking, therefore, the upper leg is lifted high enough to allow the dependent foot to be swung clear of the ground, and this accentuated flexion of the thigh produces what is known as "steppage gait." If the lesion is in the upper motor neuron we have weakness, but not actual paralysis in the limb affected until a late period of the disease when the patient is helpless, and the variety of gait cannot be a factor in diagnosis. Since any lesion in the upper motor neuron releases the spinal reflexes from the inhibitory control of the brain, the tendon reflexes are all exaggerated, and the limb becomes stiff (spastic). Since the trophic centers lie somewhere in the gray matter of the cord, and not in the lateral tracts, no atrophy results, except such as would be expected to arise from disuse. Two classes of cases are found in this category, viz., those arising from lesions of the tracts, and those from lesions in the brain. In the first case the stiffness of the limb makes the complicated process of walking unusually difficult, since that action is properly performed only when we are capable of instantaneously making numerous slight muscular readjustments at every stage of the process. This is further complicated by the fact that the limb is weak. In the study of the physiology of the cord it was stated that the stretching of a muscle when inhibition was removed from the cord would produce clonus, and we therefore have a weak limb which is stiff, and the muscles of which are the seat of possible clonus. All these factors coöperate in producing the peculiarities of the spastic gait. The leg being stiff tends to scrape along the ground "en masse," and to avoid this as far as possible the pelvis is tilted up on that side, the leg is swung forward as one piece; since it is weak the step is short, and the propelling leg is the seat of clonus, as soon as the muscles are put upon the stretch. This makes the posture of the person uncertain and teetery; this is called "titubation." Observe for future reference that the steps are placed in a straight line, and that they are short. In the second case, where the cortex is the site of the lesion, the adductor muscles in the thighs are spastic to a variable extent, sometimes to such a degree that the knees rub together, or so much that the legs cross, and we get "cross-legged" progression, or "scissors-leg." If now the lesion which has produced a pathological gait is not in either the upper or lower motor neuron, but is in the posterior columns of the cord, or the direct cerebellar tracts, or cells of the column of Clarke, we find an ataxic gait. There has been an injury of the fibres which convey sensory impressions to the reflex arc, or data to the cerebellum as to the tension on tendon and muscles, or pressure upon joint-surfaces. We stand readily or walk correctly from the ability to constantly readjust our position by the information conveyed to us from sensory impressions from the soles of our feet, and knowledge of the pressure and tension that is being exerted. The results of such losses are these: the muscles are not paralyzed, but force is exerted without proper direction unless guided by vision; the leg is lifted extravagantly, and the foot is projected without accuracy of direction, or to an equal extent in successive steps; it is not set down with precision, but is slapped down, the heel usually striking first. The result is that the person sways from side to side, the steps are of irregular length, and are not in a straight line. The feet are widely separated, both in walking, and in standing, and walking backward accentuates the disability.

SCHEME OF EXAMINATION OF CASES OF NERVOUS DISEASE.

Name—Sex—Age—Civil Condition—Married—Single—Widow or Widower—Resides—Date.

FAMILY HISTORY. Father. Mother. Condition of their health and of their ages, and their history in relation to habits and diseases. If either or both are dead, find age at death, and cause of death. Number of brothers and sisters, and the patient's place in respect to the age of the others. Their state of health, ages, and if dead, cause, and age at death.

PERSONAL HISTORY OF PATIENT. Habits, present and past. If married, the state of health of the husband or wife, the number and health of the children, and miscarriages, if any, and their relation to living children. Patient's manner of birth. His health as an infant, convulsions if any, and the cause assigned for them. Diseases of infancy, and their severity, and after-effects. Diseases of childhood with their grade of severity and after-effects. Diseases of adult life with particulars (venereal especially). Injuries suffered.

PRESENT ILLNESS. When symptoms first appeared, and what they were. Order of onset, rate, and if preceded or accompanied by fever or pain, and what cause was assigned for the onset. Present symptoms complained of. This includes all disabilities or abnormal sensations, or sphincter disturbances which the patient is able to note. (These are subjective symptoms, if they are such as can only be perceived by the patient.)

EXAMINATION. General condition of nutrition, and size and weight. Marks of injuries or diseases.

Manifest marks of degeneration.

Firmness of muscles, and atrophies, or hypertrophies.

Ability to perform movements.

Possibility of passive movements.

Movements of eyes:
  Hemianopsia.
  Extent of visual field.
  Extent and anomalies of color field.

Movements of face.

Movements of tongue.

DEEP REFLEXES:
  Knee-jerk.
  Achilles-jerk.
  Ankle-clonus.
  Plantar reflex.
  Babinski's reflex.
  Gordon's reflex.
  Oppenheim's reflex.
  Wrist-jerk.
  Jaw-jerk.
  Malar-jerk.

SUPERFICIAL REFLEXES:
  Cremasteric reflex.
  Hypogastric reflex.
  Epigastric reflex.
  Pharyngeal reflex.
  Corneal reflex.

LOOK FOR:
  Argyll-Robertson pupil.
  Romberg's sign.
  Kernig's sign.
  Hypotonia.
  Asteriognosis.
  Adiadochokinesis.
  Tremor.
  Incoördination.
  Anesthesia.
  Hyperesthesia.
  Analgesia.

Thermesthesia.
(These are objective symptoms.)
Orientation. (Objective and subjective.)
Sphincter disturbances. (Organic reflexes.)
Tics.
Habit spasms.

MENTALITY:
Delusions.
Illusions.
Hallucinations.
Aphasia. What variety and extent.

The methods by which the definite senses are examined are stated in detail in the description of the various conditions in which they are found to be perverted, and it is necessary here to speak only of general methods of investigation. Tests for the cutaneous sensibility may be made by ascertaining whether or not the patient feels the touch of a camel's-hair brush, or of a little pledget of cotton, or still better if the light touch of the examiner's finger is perceived. The latter is the better instrument, as it is always accessible, and with a little practice the touch may be very delicate, and the relative sensitiveness of various areas is capable of very accurate estimation. Æsthesiometers of many varieties are for sale at the instrument-makers, but are not usually a necessity. The perception of pain may be investigated by pricking the surface in various areas with a common pin, and this instrument is better than a needle, since the latter may be so sharp that its entrance is comparatively painless. The perception of temperature is to be tested by the alternate use of a test-tube filled with cracked ice, and another filled with hot water. Care must be used that the cold tube is kept dry on the surface, else tactile sense may discern the moist surface of the one as opposed to the dry surface of the other. In the prosecution of all these tests the eyes should be carefully covered, and the site of the tests should be abruptly changed, and not only the kind of sensation, but its localization should be required of the patient. Response should be prompt, since delay of perception is a grade of loss. Remember that in temperature investigations a patient may lose the perception of heat, and not of cold, and vice versa. I have found it necessary to warn patients that no restraint is demanded of them, for in some cases the tests for pain gave at first confusing results, as the patient thought it the part of valor to avoid any symptoms of distress as long as possible. Tremor is tested by showing the teeth with as much rigidity of the lips as possible; by opening the mouth and thrusting out the tongue as far as possible, while at the same time the arms are stretched to their full extent above the head; by stretching out the arms at full length in front of the body, and at the same time separating the fingers. A more delicate test of tremor of the hands is by letting the tips of the outstretched fingers lightly press against the palm of the hand of the examiner. A very faint and fine tremor is discernible by this method. Handwriting will show tremor and ataxia also. The detection of passive and intention tremors is described in detail in the articles upon paralysis agitans and multiple sclerosis respectively. Ataxia and incoördination are described in full in the article on tabes dorsalis. As a basis for diagnosis, the first question to be decided is this: Is the disease so sudden in onset that it must have resulted from traumatism (hemorrhage for instance), or gradual enough to allow an inflammatory process to be the cause, or has it been slow enough to have been the result of a degeneration? Is the locality of the lesion in the peripheral nerves, or is it central, i. e., in the spinal cord, or brain? If it is located in the central nervous system, is it upper or lower neuron in type?

Spinal cord affections may be arranged in three groups:

1st. Those in which the symptoms come on all at once.

2nd. Those in which the symptoms are acute, taking several hours, or even days to develope.

3rd. Those in which the symptoms are insidious.

When the symptoms begin all at once, there are but two lesions to think of: tissue-trauma, and hemorrhage. If it is traumatic, there is the history of evidence of injury, usually fracture, or fracture-dislocation. If it is hemorrhagic, the cord has already been softened by myelitis, or a new growth, or the person is syphilitic, or possibly has anemia.

THE THERAPEUTICS OF NERVOUS DISEASES.

The therapeutics of diseases of the nervous system differ from those of the body in general for several reasons. The medicinal treatment of the diseases of the rest of the system has been a slow accretion of the experience of the centuries in its quest for a healing agent for pathological changes in material structures which have been more or less possible of demonstration to the senses, or have been so readily influenced by remedies that numerous experiments have been possible, and the results have been unequivocal. Nervous diseases have not rested upon changes which were palpable or appreciable; some of them have not even yet yielded their secret to our more refined methods of investigation. They, in general, are not self-limited, and some of them take years for their full developement, and hardly one of them is curatively influenced by any kind of medication with enough rapidity to make the results striking, or even appreciable, except by observation for a long period. It may be more explicitly stated that until within a very few years, the causes and the seat of the characteristic pathological changes pertaining to any special disease were not known at all, and some are still shrouded in the same obscurity. All schools of medicine were therefore unable to furnish much material for our therapeutic guidance, since they were generally in no position to identify, or to rightly interpret the significance of the symptoms concerning which they were making their reports, and which they were assuming to have been relieved by their medication. This statement is written in full knowledge of the claims of both schools, and of the therapeutic accumulations of the years before even a few of these diseases could be diagnosed with any degree of accuracy. We may own that many a physician has cured a complaint which he could not diagnose, but we cannot accept his therapeutic conclusions, when we are certain that he was ignorant of the identity of the symptom-complex for which he announced the discovery of a remedy. We may perhaps infer that his description refers to the disease which we have under consideration, but we must repeatedly obtain similar results in order that his statements may carry conviction. Therefore it may be safely affirmed, no matter what may have been the school of medicine, or what may have been the nationality of the writer, that any remedy recommended over thirty years ago, for any given nervous disease, should carry very little weight, for no one at that time could know either the etiology, pathology, or symptomatology of the disease for which he makes the recommendation. When one looks over the chronology of the discovery and primary isolation of the actual changes characteristic of the most common diseases of the nervous system he is struck by their recent date, since 1868, 1870, 1876, and so on, are the rule, and , as I have said before, many of them still hold the secret of their cause and its pathological expression. Who can certainly tell the cause of epilepsy, of chorea, or hysteria?

In many cases therefore symptomatology is the only possible basis for prescription and, comparatively speaking, is the only scientific method. The validity of any method however is nullified in organic diseases by the fact that while nerve-fibres may be regenerated, nerve-cells once destroyed are never replaced by tissue capable of nervous function. If therefore we have grounds for belief that the disease has destroyed nerve-cells, we know that medicine, however skilfully selected, and by whatever method, can do no more than stimulate the remaining cells to their highest grade of function. All claims beyond this are a cruelty to the confiding patient. In functional diseases we have grounds for great confidence in our therapeutic measures, but we should make ourselves sure by a diligent study of the pathology of any condition in question that we are not confusing remission with cure.

CHAPTER III.

PERIPHERAL NERVES.

ANATOMY. Cranial and spinal nerves are constituted in a similar manner, except that some cranial nerves are either solely motor, or solely sensory in function, most of them being both sensory and motor, while the spinal nerves without exception are mixed nerves. The fibres of the motor part arise from the cells in the anterior horns of the spinal cord, while those of the sensory find their nutritive center in the cells of the ganglia which are located on the pedicles of the vertebræ. The union of the two parts takes place just outside the ganglion. The cranial nerves get their motor fibres from nuclei in the medulla and pons and crus, which are the analogues of the cell-aggregates in the anterior horns of the cord, while the sensory fibres have a common origin in the widely-distributed nuclei of the fifth nerve. In all cases the emerging nerve-fibres are covered by prolongations of the pia and dura until the union of the motor and sensory roots takes place just outside the root ganglion, when the completed nerve is invested with a thick layer of lamellated connective tissue, known as the epineurium.

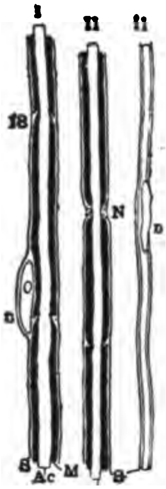


FIGURE 30. EXHIBITS THE STRUCTURE OF NERVE-FIBRES (FROM GOWERS).

I and *II* show a medullated, and *III* a non-medullated fibre. *Ac*, is the axis-cylinder or neuraxon; *M*, is the envelope of myelin; *S*, is the connective-tissue sheath, or sheath of Schwann; *n*, is the nutrient cell of this sheath; *IS*, is the incisure of Schmidt and Lantermann; *N*, is a node of Ranvier. The myelin appears black from the osmic acid stain.

HISTOLOGY. A nerve, as we see it, is a mass of mixed tissue designed for the protection and nutrition of the nerve-fibres in their distribution to the structures of the body, and its essential part is the nerve-fibre which we first centrally discover as a filament emerging from a nerve-cell. While it is not now considered certain that it originates in that cell, we do know that its existence and functional ability depend upon the integrity of the cell. The nerve-fibre or axis-cylinder is surrounded by a white, semi-fluid substance which is called the medullary sheath, the white substance of Schwann, or myelin. The form and consistency of this white substance is imparted to it by a net work of a fine horny substance called neuro-keratin. Myelin is absent from the structure of the sympathetic or non-medullated nerves. About this myelin is a delicate membrane which is termed the primitive sheath, neurilemma, or sheath of Schwann. Nuclei lie between the myelin and the primitive sheath, one cell to each division of this structure, which divisions are marked off by constrictions by which the myelin coating is interrupted, these constrictions occurring at distances of about seventy-five times the diameter of the medullated nerve-fibre.

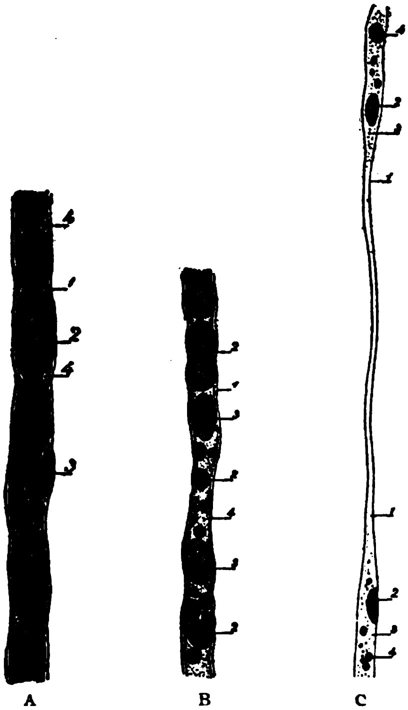


FIGURE 31. DEGENERATION OF NERVE FIBRE.

Nerve tubules in peripheral portion of a divided nerve—A, two days after section; B, four days after section; C, eight days after section. 1, Sheath of Schwann; 2, myelin; 3, nuclei; 4, protoplasm (Ranvier). (Church & Peterson. Courtesy of W. B. Saunders Co.)

These divisions are called the nodes of Ranvier, and each internodal section in its growth and degeneration, acts as a unit, and may be considered a single structure with its vital part represented by the cell. In addition there are incomplete divisions, concerned in some way with nutrition, which are called the incisures of Schmidt. The function of the myelin may ensue the conductivity of the nerve-fibre, as does the insulating material in an electric cable, and may also be nutritive, but these functions are as yet only conjectural. All spinal nerves contain some sympathetic fibres, but they are absent from the nerves of special sense, except in the case of the olfactory. The nerve-fibres with their coating of myelin are assembled into bundles which are covered by a nucleated envelope of connective-tissue, which is called the endoneurium; larger bundles are composed of numbers of these small fasciculi and covered by another envelope of connective-tissue called the perineurium, and these finally are aggregated into what we know as a nerve by a covering called the epineurium. All these connective-tissue envelopes are continuous, and are ramified by blood-vessels and lymphatics, and the nerves of the nerves (nervi-nervorum) which are derived from the nerve-fibres which they supply.

PHYSIOLOGY AND PATHOLOGY. The nutrition of the fibre depends upon the integrity of the cell, and impulses seem to come from it, but not solely, since excitation of fibres can inaugurate impulses. While there are cells in the envelopes there are none in the fibres, and therefore it is plain that nutrition must come from a cell elsewhere. In addition to nutrition, the cell has some function in the transmission of impulses to and from the brain. Sensory fibres have wide expansions at the periphery, so as to gather stimuli from the widest possible area, but motor cells have their endings in definite and minute primitive particles of tissue. Protection of the nerve-fibre by the vitality of its envelopes seems to be a necessity; normally it is covered by neurilemma and myelin; in some places the myelin is lacking, and the neurilemma alone persists. If in either of these situations the neurilemma is injured the nerve immediately begins to degenerate. The portion in connection with the brain or cord retains its normal condition, with the exception of a few millimeters at its peripheral end, since this portion of

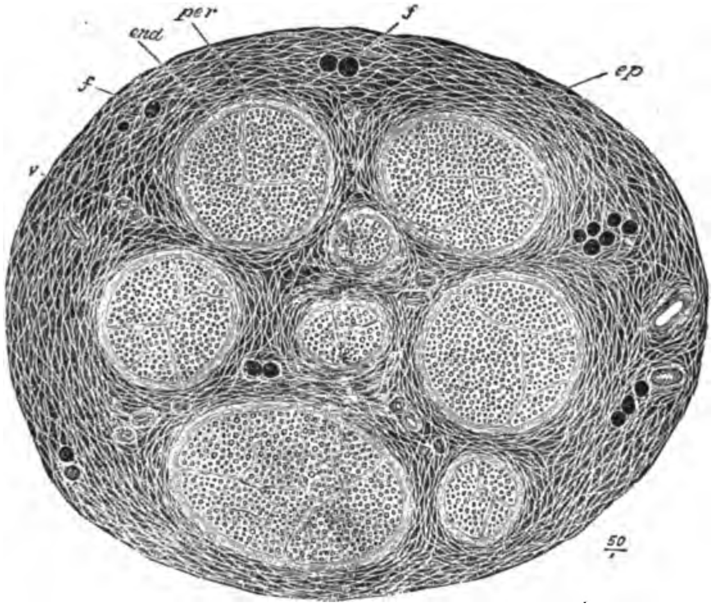


FIGURE 32. Transverse Section of the Internal Saphenous Nerve of Man, made after being stained in osmic acid, and subsequently hardened in alcohol. Drawn as seen under very low magnifying power. Showing ep, epineurium or the general sheath of the nerve, consisting of connective tissue bundles of variable size, separated by cleft-like areolae, which appear as a net-work of clear lines, with here and there fat-cells, f, and blood-vessels, v; per, perineurium, the lamellated connective tissue, enclosing the funiculus; end, interior of the funiculus, showing the cut ends of the medullated nerve fibres which are imbedded in the connective tissue within the funiculus (endoneurium). The fat cells and the nerve fibres are darkly stained, but the connective tissue of the nerve is only slightly stained. (Quain.) Courtesy of Wm. Wood & Co.

the nerve is still connected with its nutrient center, i. e., the cell in the brain or cord. In degeneration the first change is a primary degeneration of the myelin; axis-cylinders and nerve cells are evidently affected later. Degeneration is in the direction of conduction in motor fibres, i. e., toward the periphery, but is contrary in sensory fibres, in their peripheral parts, since their nutrient cells are central to the fibres, but in the cord the rule holds true, since the cell is here peripheral to the fibre. Degeneration is usually secondary, since it generally arises from a primary lesion like the division of a nerve, but there are instances, however, where it is primary, as when disease has its initial site in the nutrient cell. Secondary degeneration, also called Wallerian degeneration, is the form which is now under consideration. It is not a mere degeneration, but a method of activity of the cell of the node which has apparently been released from some unknown inhibitory influence, and takes on such an exuberant growth, that as in other new growths, fatty degeneration ensues, and destruction of the tissue is a final result. The medullary sheath is broken into fragments within twenty-four hours, and the break-down of the tissue is complete within three days, and interruption of nerve-conduction is then evident for the first time. This process is quite complete by the end of the fourth day, and then these small fragments become converted into fat globules, a process of saponification ensues, and the substance passes out through the neurilemma, and is removed by the lymphatics, so that by the end of the third week the neurilemma is an empty sheath, with the possible exception of some finely granular deposit. In central lesions this process begins simultaneously at several points, but in peripheral nerves it proceeds from the center toward the periphery, except in the rare peri-axillary type of Gombault, seen in poisoning by lead and alcohol, where degeneration takes place from numerous centers. Simple division of a nerve seems to be less serious in its consequences than an extensive dislocation of myelin by a contusion. All severe changes produce degeneration of the end-organs in the muscles, and in the case of motor-nerves produce nutritional changes in the muscles. The muscle elements waste, while there is connective tissue proliferation, which produces the contracted, board-like masses which replace the normal flexible muscular tissue. When a nerve-fibre degenerates the nerve cell is always affected to some extent.

A histological study of the nerve-fibres after separation shows that during the process of degeneration a regenerative process has been inaugurated, consisting of a multiplication of the nuclei lying just beneath the neurilemma, and an accumulation about them of a granular protoplasm, which in due time completely fills the neurilemma. At this stage it is called a "band-fibre." If the conditions are favorable the nerve-fibre grows into this band-fibre, the protoplasm becomes changed into myelin, and the axis-cylinder continues to grow forward until it reaches its original destination by insinuating itself into the old sheaths, and so on, finally elaborating the appropriate end-organs anew.

Another method is described thus: there is a proliferation of the neurilemma cells, and spider-like neuroblasts are seen sending out beaded axis-cylinder processes in both directions from opposite poles. At the end of the fourth week these processes may overlap and anastomose. A new medullary sheath appears about the eleventh week. The regenerative process begins about the third week in experimental cases. The process described indicated that while regeneration takes place from the central stump, and has been held to be the fact in all cases, that there is now some evidence that the process may depend upon the activity of neuroplastic cells remaining in the granular detritus, seen in the shrivelled sheaths of Schwann, and therefore regeneration is simultaneously inaugurated in every segment, just as bone is reconstituted. It is probable that processes differ according to the different stage of degeneration at which the regenerative process begins. Neither axis-cylinder nor medullary sheath attains its full size until it unites with the stump from the cell. Unmyelinated fibres, the conduction tracts in the brain for instance, never regenerate since they have not the necessary cell elements to accomplish the process. Regeneration is a slow process, taking from two to four months in a short nerve, while in the sciatic it may take from one to two years. Nerve conduction, and the possibility of excitation do not always progress at the same rate.

NEURITIS.

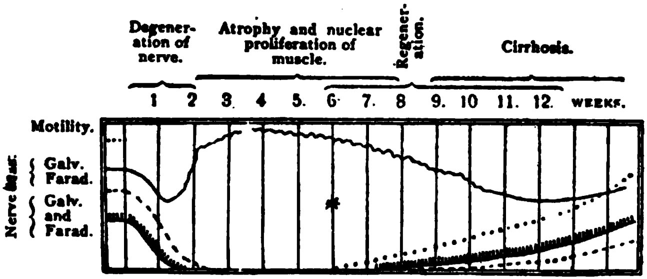
DEFINITION. This is an inflammation of a nerve or nerve-trunk, or the degeneration of nerve-fibres. The resulting symptoms of the interstitial forms are the effects of a compression of the nerve-fibres in the nerve-trunk by an inflammation of the connective-tissue envelopes, rather than of the fibres themselves.

DIFFERENTIAL DIAGNOSIS. It should be differentiated from neuralgia and hysteria.

ETIOLOGY. Neuritis may arise from infections, chronic intoxications, and the various cachexias. Refrigeration has been given as a competent cause, as has privation, yet it is probable that they do not often act directly, but only by so reducing the resistance of the individual that he falls a victim to influences which might have been successfully resisted under better conditions. It has been seriously questioned whether such a condition as ascending neuritis actually exists except as the result of an actual infection of a nerve-trunk, but it seems to have resulted from appendicitis, and from pneumococcic invasions of other structures. Many nutritional diseases have been observed to be marked by paresthesias, stiffness, and soreness of the muscles, which symptoms have proved to be the signals that the delicate nervous structures were suffering damage. A disseminated neuritis has been the inaugural stage of arthritis deformans, and it has been proved that disease-processes in nerves usually affect the cell in the cord to some extent.

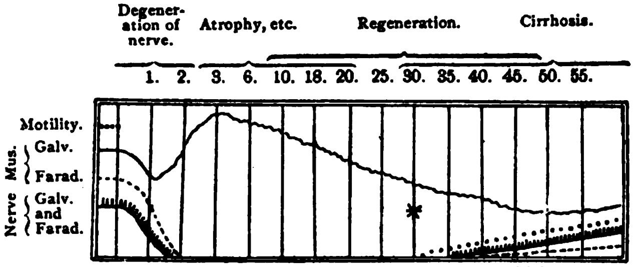
PATHOLOGY. The inflammation may be in the perineurium, or be a general interstitial process, or be parenchymatous, i. e., in the nerve-fibre itself, and in all cases the different structures participate to some degree. It may be localized in a small section, or be disseminated over a large area, or affect the whole length of a nerve, or affect many nerves simultaneously, or involve them by a gradual extension, as in multiple neuritis. Constitutional states or chronic infections are apt to produce the last form. A perineuritis, or an interstitial neuritis, is an inflammation while the parenchymatous form is a degeneration. There is little difference in their symptomatology. Where there is an inflammation of a nerve, its envelopes lose their lustre, and there is swelling and redness of the connective tissue. If a section is examined with a microscope we find that the blood-vessels are congested, and there may be signs of small hemorrhages; there is an effusion of white cells, and there is to some extent a proliferation of the connective tissue. This process affects the whole nerve, if long continued. In the interstitial cases the process is the same, but begins in the connective-tissue envelopes of the subdivisions of the nerve, and almost certainly affects to a degree the fibres which are less often and less seriously compromised in the epineural type. This interstitial form is most certain to set up proliferation of the connective tissue, and to produce a swelling of the nerve, which is apt to make such a section spindle-shaped. The parenchymatous form is the most common, and it may be primary or secondary to the two preceding, or arises from injury to the nerve-trunk. It has been thought that all non-traumatic neuritis arose from primary affections of the nerve-cells, although perhaps so slight as to be undiscoverable with our present means of investigation, but that is not as yet certain.

SYMPTOMATOLOGY. Where a single nerve is involved, the advent may be unmarked by any constitutional symptoms, but where several nerves are simultaneously attacked there are generally fever and chills, which may be repeated if the process invades new areas with a great degree of violence.



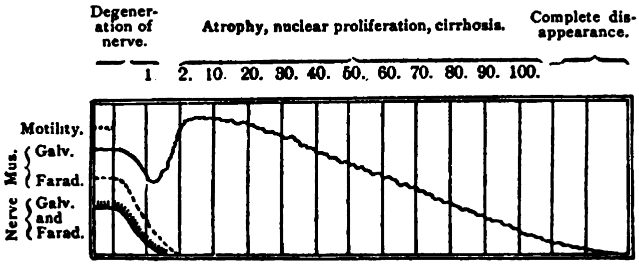
33

The first ordinate shows occurrence of the lesion and cessation of motility; the star indicates the return of the latter, the waved line indicates qualitative change



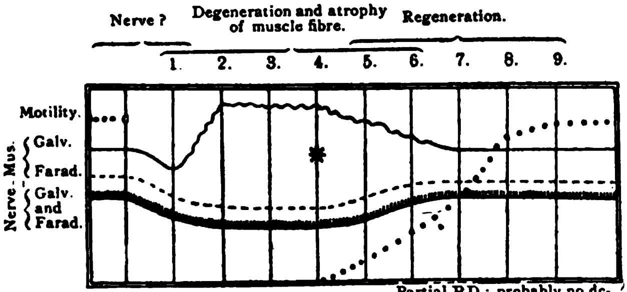
34

Paralysis with late return of motility.



35

Incurable paralysis; no return of motility.



36

Partial RD; probably no degeneration of nerve.

FIGURES 33, 34, 35, 36. (From O'Connor.)

The constant symptom is pain, severe, boring, tearing or burning, which may be localized or diffused, and last for weeks or months, but with some variation of intensity. This pain is increased by anything which places pressure upon the inflamed structure, or which puts tension upon it, and it is not merely local, but radiates over the whole distribution of the nerve. A nerve is peculiarly susceptible to pressure at the points where it emerges from a bony canal, or winds about a bone. The swelling may not be conspicuous, but on the other hand it may enlarge the nerve to twice its normal size. The process, it is seen, is not different from that common to all structures of a similar histology, but the results are colored by the fact that structures conveying sensory impressions are involved in it. The symptoms most suggestive are the paresthesias and hyperesthesias in regions remote from the palpable symptoms, but known to be innervated from the nerves in question. This relates to the sensory fibres, but irritation of motor fibres may set up fibrillation, muscular contractions, and an increase of myotatic response. In the first stages tendon-reflexes may be increased, but the violence of the attack may diminish them at once, since the final outcome in all cases is a diminution and final extinction of these reactions. Electrical excitability is increased at first, but it is finally reduced or entirely abolished, so soon as there is a more or less complete break in the continuity of the nerve-fibre. (1st) There is a sharp increase in the response to both faradism and galvanism in the muscle. (2d) After the second day there is a rapid decrease of response to both faradic and galvanic currents up to the tenth day, when it is completely lost. (3d) In the second week, faradism being absent, there is such an increase of response to galvanism that the usual degree of reaction can be invoked by currents of one twentieth the usual strength. (4th) If regeneration takes place, its presence is marked by a decrease of this over-excitability of the muscle to galvanism, and a gradual return of the faradic irritability. The degree of damage to a nerve is determined by the degree of this so-called reaction of degeneration. (5th) If regeneration is not accomplished, the galvanic reaction decreases to apparent obliteration in from three to nine weeks, but actually a sluggish response to the anodal (positive) closure may be discovered for a year or more, if the current is very strong. In addition to this change to quantity there is also a qualitative one, in that while normally muscles react more strongly to the negative (cathodal) closure, in the reaction of degeneration the positive is the more potent. The foregoing refers to the reaction of muscles influenced through the nerves at the so-called motor points. When we apply the electrical current to a nerve-trunk, at some accessible point, its degeneration can be discovered by the fact that it loses its excitability quanitatively only, there being no inversion of the customary response to either pole, and faradism and galvanism both lose their power. There is a slight modal change in the early stages, in that the response is sluggish instead of being prompt.

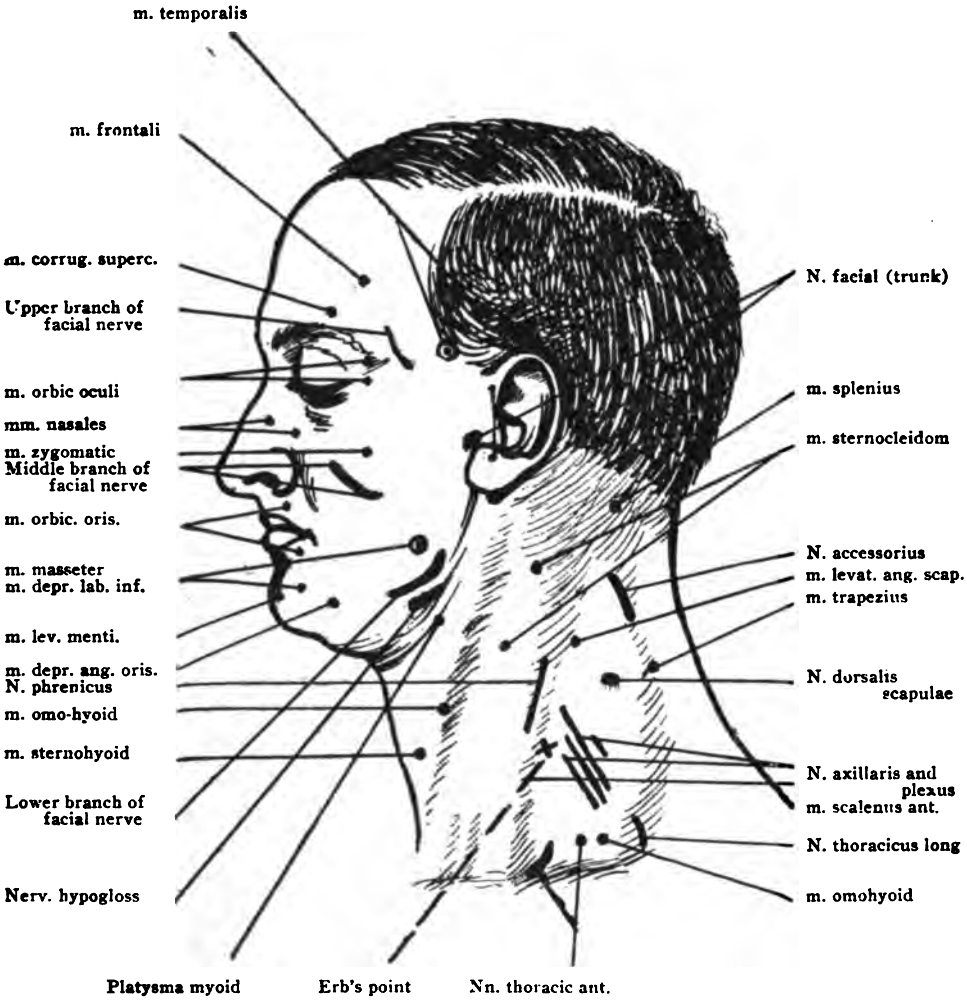


FIGURE 37. (From King.)

Trophic disorders, shown by glossy skin, various eruptions, joint-changes, but most of all by atrophy of the muscular masses, are always present to some degree, unless the case is one purely epineural, or where the process is so short that recovery antedates such changes. In such cases we know that the fibres have not been involved, or have been only functionally disturbed.

COURSE AND PROGNOSIS. The disease tends to be a chronic one, but may terminate in a few weeks. Some cases endure for years, and not infrequently for six months to one or two years. The cases which gradually develope in a manner suggesting chronicity are apt to last longer than those which are acute from the first, and those from rheumatic, or toxic, or infectious causes have also a relatively good prognosis. In the interstitial and parenchymatous types recovery is apt to be imperfect, and at the best very long delayed. The possibility of ascent to the cord from a primary peripheral site is not as common as was formerly held, and is practically true only of those cases where the nerve is the seat of an active infection.

DIAGNOSIS. We base our diagnosis upon the presence of symptoms along some definite nerve-trunk, and if the tenderness is not confined to the nerve, if the sensory disturbances and loss of power do not conform to the distribution of this nerve, and if the appropriate tendon reflexes are not lost, it may safely be assumed that the case is not one of neuritis. As has been said, temperature may show little or no disturbance; and there is not usually much swelling or redness of the tissues about the nerve-trunk or its distribution. The sensitiveness to pressure is liable to be only slightly developed in the epineural and interstitial types. Parenchymatous or degenerative cases present less symptoms of their true nature than do the other types, since the symptoms are simply those of a break in continuity of the nerve-fibre. In some cases neuritis may be systematized, i. e., affect exclusively the motor, or, in other cases, the sensory fibres only.

DIFFERENTIAL DIAGNOSIS. See Neuralgia, and Hysteria.

TREATMENT. This will be more particularly specified when the separate types are described, but from first to last, fresh air, baths, and a very nutritious diet should be insisted upon, and speaking generally we may say that the treatment is rest, immobilization of the parts, (and this is far more essential in the milder form of cases than is generally supposed), and local applications of heat, cold, and electricity.

King says that nothing is so temporarily efficient as heat, that radiant heat (500 candle power incandescent light for instance), is far more efficient than poultices, hot water-bags, and such means, and he says that there is a variation in the value of different parts of the spectrum, the yellow being the most efficient. The amount of radiant energy applied is important. It is just as possible to make too great and prolonged an application as it is not to make application enough. When this radiant energy is applied, there first comes a blush to the surface. If the application is now discontinued this will soon disappear. The application should therefore be continued until there is a deep redness over the whole surface of the member. At this point it should be discontinued, or the bright red color will change to a darkened hue, showing that a venous congestion is taking place, and this should always be avoided.

It has usually been the opinion that the use of electricity should be deferred until the second stage, or fastigium, and the period of defervescence, but King advises, during the stage of invasion, a descending galvanic current down the nerve, increased to 10-15 m.a., and gradually decreased through a period of about fifteen minutes. In the fastigium he uses radiant heat still, but in place of galvanism advises high frequency with wet moistened and soaped tin-foil electrodes, with smooth spark-gaps, a frequency of at least two million per second, and a treatment of twenty to thirty minutes. Galvanism is better than an imperfect high frequency. For the beginning of the third stage heat should be discontinued, but use labile negative galvanism for the whole affected area with the positive on the origin of the affected nerve. Later the sinusoidal current should be employed, for its especial value is increasing conductivity in the nerve, and improving metabolism. The current should be strong enough to produce gentle contractions with each cycle of the current, which should be not more than twenty to fifty per minute. It may be useful in the early stages, but at this stage the current should never be strong.

Static affects the conductivity of a nerve when other electric modalities are not efficient. Another method of stimulation is by the so-called Morton wave current. Place a positive electrode three and a half by eight inches over the affected area, ground the negative pole, and to the patient, sitting upon an insulated stool, give the treatment with a spark-gap of from eight to ten inches, with a slow interruption for about ten minutes. Fulguration with the X-ray has benefited some cases.

The actual cautery carries this process to a yet higher grade, and has a great value in combatting the pains of neuritis. The best method is by touching point by point, rather than by stroking, until the surface turns a rich brown (Gibney).

Vibration over the points of emergence of the spinal roots produces a high degree of stimulation, and benefits such cases as are able to develope the requisite reaction.

Massage is good at all stages for the relief of pain, except where there is great swelling of the nerve-trunk. It must be very gentle in the early stages, but in the third stage should increase in severity, and should be combined with passive movements of the limbs for the cure of contractures, deformities, and atrophies. Nerve-stretching and transplantation, and sometimes resection are also indicated in obstinate cases.

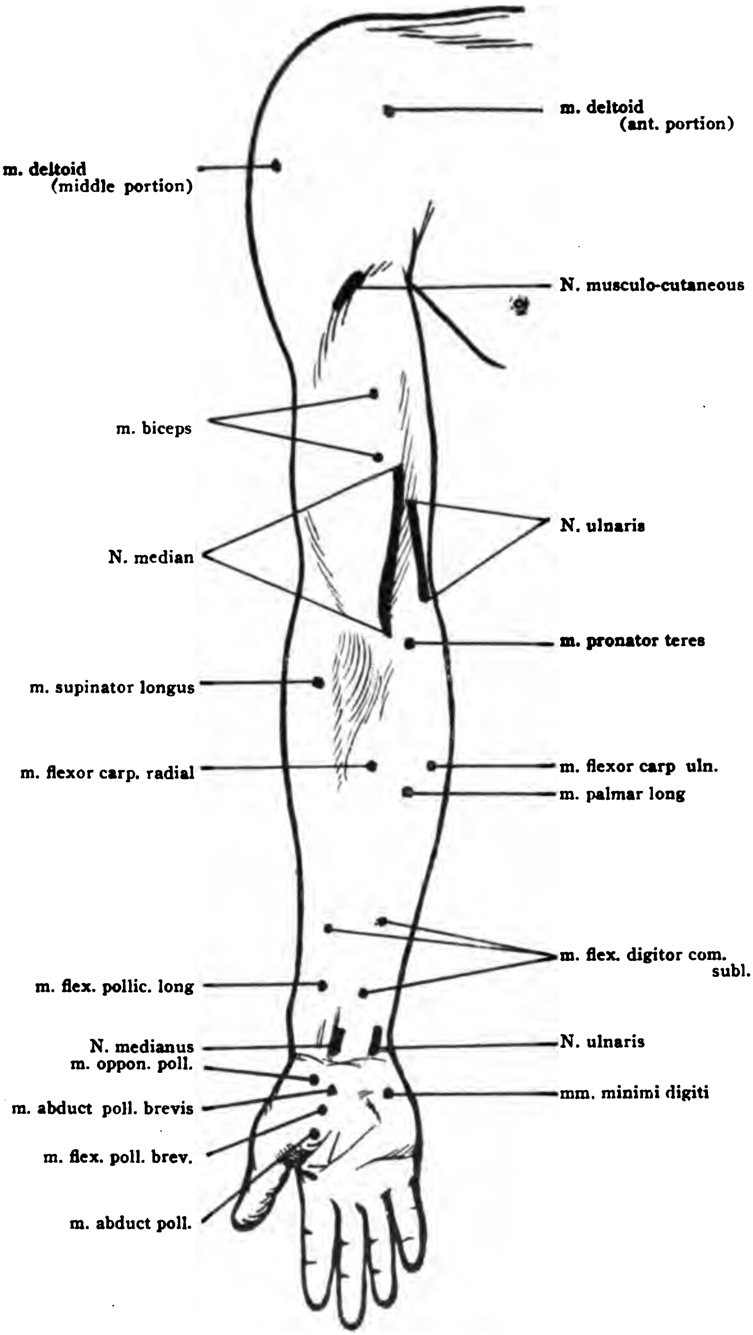


FIGURE 38. (From King.)

THERAPEUTICS. The remedies possible of application to this condition are so numerous that differentiation may be exceedingly difficult, but when we keep in mind that symptoms have a comparative value, we shall probably attain the greatest success if we diligently seek for the cause, and inaugurate our therapeusis on causal lines. If we find that a case is plainly rheumatic, we should think of Bryonia, Rhus tox., Cimicifuga, Apis, or some cognate remedy, while if the case is alcoholic we should employ Nux vomica, Cimicifuga, or Strychnia. I advise this pathological or etiological method for the reason that it is notoriously difficult to obtain data from persons who are in pain, but the fact that our first prescriptions are upon broad grounds does not indicate that as soon as we are in a position to obtain definite data we should not keep our eyes open for the faintest points of differentiation, and from this care I have seen some most dramatic cures.

Aconite. This is to be used in the earliest stages, especially if the attack has been the result of exposure to dry cold. The pain is tearing, with nightly aggravation, and mental anguish. It is never applicable unless numbness and tingling of the affected parts is present.

Anantherum muricatum for root-neuritis (Dewey).

Arnica. Cases resulting from crushing and bruising injuries.

Arsenicum. This is probably the best remedy for the severe type of cases, and always to be thought of in neurasthenic persons. There is a great intolerance of conditions, and a generalized irritability. It often originates in a traumatism of a nerve, and there is exquisite tenderness to touch in the whole limb. In proper cases it will relieve where opiates have failed. There is severe burning, and also in those cases where there is paralysis, relieved by hot applications and pressure, and the attacks are liable to be paroxysmal at night.

Argentum nit. This is of value in cases from acute infections where the patient becomes ataxic.

Belladonna. In cases where the pain is atrocious and unbearable, but the case is sthenic instead of neurasthenic, as in the Arsenic cases.

Bellis perennis. This is for bruised pains, similar to those of Arnica, but they are the result of chilling after being overheated. It is a pronouncedly left-sided remedy.

Bisulphide of Carbon has been suggested as a remedy in the ataxic cases resulting from acute infections.

Carburetum sulph. Valuable in neuritis, especially in the sciatic variety (Dewey).

Causticum is suggested for an ataxic type resulting from infections.

Cimicifuga. For a neuritis, conspicuously that from alcohol, marked by a tenderness of the nerves, rather than by spontaneous pain. When there is pain it is like toothache in its character. O'Connor advises one drop of the tincture in four ounces of water, a teaspoonful of the solution given hourly. Dewey recommends Ledum and Plumbum for this form of neuritis.

Conium mac. This is advised for the later stages of a traumatic neuritis.

Gelsemium. The principal remedy for the acute form of neuritis from infectious diseases, if paralytic in type, and a valuable remedy on very general indications.

Hypericum. A traumatic remedy like Arnica and Conium, but rather for the lacerated type of injury than for the bruised. It has proved useful to the author in injuries of all grades, from an injury to small peripheral branches, up to severe traumatism to an entire plexus. The best results have been seen when the 30th potency was employed.

Nux vomica. This is useful in the 1st potency in alcoholic cases. The pains are intermittent, commence in the morning, are violent, excruciating, sharp and short, lancinating with jerking; often worse from pressure and warmth.

Potassium iodide. This is said by Bartlett to be of value in the neuritis of old people, in doses of five grains, three times a day.

Phosphorus where the nerve has degenerated.

Plumbum is to be given when atrophies are found.

Rhus tox. is applicable to more cases than any other remedy, peculiarly so to those caused by exposure to damp cold. They are the rheumatic cases, in which the coverings of the nerve-fibres are affected. They are the interstitial and perineural types. To be thought of in cases from acute. infections also, and in paralytic cases after the subsidence of inflammatory symptoms. The pains are ameliorated by motion, and aggravated by cold, dampness, and the long maintenance of one position. Bartlett advises the employment of liquid preparations only, and favors a dose of one drop of the first dilution every hour. Dewey states that Elliot believes that Ferrum phos. and Kali mur. are the preferable remedies for rheumatic neuritis.

Strychnia sulph. When the active symptoms have subsided, and there is great prostration.

Thallium for root-neuritis (Dewey).

Asparin. For the relief of acute pain Asparin is one of the best of physiological remedies. It may be given in doses of from five to ten grains, from three to six times a day, according to the size of the dose, preferably in lemon water, or in milk.

MULTIPLE NEURITIS.

POLYNEURITIS, PERIPHERAL NEURITIS, SYMMETRICAL NEURITIS.

DEFINITION. This is a condition where many nerves are attacked in rapid succession by a degeneration of their axis-cylinders, far more than the interstitial portions, and most noticeably at their terminations in the extremities. While this process extends toward the spinal origin of these nerves, it commonly ceases before reaching the nerve-roots, but exceptionally may involve the spinal nuclei, or some of the cells of the cortex. It perhaps will finally be termed a general parenchymatous degeneration, possibly of the whole nervous system, but with a marked predilection for the peripheral portions.

THE SYMPTOMS OF A TYPICAL CASE. There is some degree of paralysis of all the extremities, with wasting, and later contraction of certain groups of muscles, especially those innervated by the musculo-spiral and anterior tibial nerves. This paralysis will be preceded by tremor and sensory changes, or accompanied by them, and whatever be the extent or distribution it will be symmetrical. The sphincters remain normal.

DIFFERENTIAL DIAGNOSIS. To be differentiated from Neuralgia, Tabes Dorsalis, Anterior Poliomyelitis acuta, Landray's Paralysis, Myelitis, Hysteria, Spinal Hemorrhage, Spinal Meningitis.

AGE. Twenty-five to thirty-five. Children furnish but a small per cent. of the cases, being immune to all causes but diphtheria, or arsenic, and occasionally malaria or measles, while old age suffers but rarely, and then from arterio-sclerosis, atheroma, and diabetes. Thirty to forty, most common age of alcoholic form. Herter details a case in a boy three years of age arising from a drink of whiskey.

SEX. About equally when all causes are considered; but from alcohol alone, the commonest cause, the female preponderance is great. Males more rheumatic and toxic forms.

ETIOLOGY. In assuming a certain causation we must remember that the disease is very often the result of several factors. In no disease is the combined potency of a predisposing and an exciting cause more noticeable. A cachexia, or a tolerable degree of toxemia or auto-intoxication, may be rendered intolerable by the added element of privation, exposure, or over-exertion. To estimate truly, therefore, the causative element to be combatted, it seems to me that these three factors just mentioned should, once and for all, be relegated to the minor rôle of exciting causes only, notwithstanding the opinion of competent observers, that at times no other cause can be discovered.

The cases fall into three classes: those due to toxic agents; those due to acute infection; and those due to a generally diseased condition of the body. In the toxic class the most potent is alcohol, its victims outnumbering all other causes combined, not from occasional excess, but from steady use of the stronger liquors. Wood alcohol is very potent. Next is lead, then far behind is arsenic, then zinc, mercury (denied by many), then phosphorus, bi-sulphide of carbon, oxide of carbon, ergot and silver. Arsenic given therapeutically has been a classical cause, but lately many protests against its potency have been recorded. It might, however, be incidentally mentioned that an Italian physician produced many cases, and of great severity, in an attempt to jugulate a group of cases of chorea by doses of Liquor Potassii Arsenit. larger than are commonly used. While it may be said that properly regulated doses of arsenic rarely have been known to produce this unfortunate sequel, in combination with moderate amounts of alcohol it is very potent. Such are the cases of an epidemic form traced to the use of beer in which arsenic, as an impurity of glucose, has been mixed. Osler mentions a case from the use of large doses of the sulphocarbolate of zinc for two years. Tobacco may be a cause.

Occupation. Plays a large rôle in the case of painters, plumbers, typesetters, rubber-workers, match-factory employees, white-lead makers, lead-miners especially, all workers in lead, arsenic, mercury, and dealers in spirituous liquors who are apt to indulge in them to excess. More than one cause may be operative, as a consumptive may become alcoholic, or physical or mental depression in an alcoholic may precipitate a neuritis.

The acute infections are potent in this order: diphtheria, smallpox, variola, typhoid, post-operative conditions from posture and narcosis (I saw a case after laparotomy), the puerperal state, puerperal septicemia, malaria, acute rheumatism, beri-beri, influenza, the exanthemata. The effect of these may be immediate, but is more often seen during convalescence, or a week or two after it; notably so in the case of diphtheria.

Conditions which deprave the general health of the body. Arteriosclerosis acts by gradually reducing the peripheral circulation; syphilis, tuberculosis, leprosy and diabetes all reduce the general health, and also specifically attack the nerves at the point where nutrition is weakest. Epidemics having the character of beri-beri have been noted in Connecticut by Hammond, and in Hamburg by Eisenlohr.

Pathology. Cases have been found with no ascertainable lesion, and the present state of our knowledge does not always enable us to determine the cause from the character of the lesion. The most usual effect is a degeneration of the axis-cylinders which is most evident at the periphery, and its intensity decreases from thence toward the cord. Wallerian degeneration is set up, and secondarily the endoneurium and perineurium are affected more or less according to the intensity of the process, with the result that in cases of long standing there is some grade of swelling of the nerve-trunk. In an ordinary neuritis the process is reversed, in that the interstitial tissue is first affected, and the axis-cylinder only as a result of the primary changes in the interstitial tissue. It might seem probable from the primary implication of the axis-cylinder at its peripheral extremity that the actual injury was to the cells of the anterior horn, and that a weakened cell displayed its debilitated condition by the death of its axon at the point where a diminution of nutrition would be most evident. In cases secondary to constitutional diseases, such as tuberculosis and leprosy, changes of a specific character have been found in the nerves. The cells of the anterior horns have shown changes in diphtheritic and alcoholic cases; the conduction tracts, both motor and sensory, and the cells of Clarke's column have been found to be degenerated. These changes are sometimes inflammatory, but generally degenerative, and a case of multiple sclerosis has been noted subsequent to a diphtheritic polyneuritis. The cellular changes are often inflammatory, and at times hemorrhagic. Diphtheria affects the brain, the cerebellum and the cord, and yet some cases, while widespread in their phenomena, have revealed no changes at the autopsy (Hosche). Lead, mercury almost solely, and alcohol, especially wood alcohol, act upon the cortical cells. Diphtheria frequently affects the ocular muscles. The optic nerve is often affected, and alcohol (wood alcohol especially) is the most frequent cause of toxic amblyopia. All the oculomotor nerves may be affected. When we find that a certain disease is symmetrical and wide-spread, we are forced to conclude that it is due to the action of some poison which is universally distributed throughout the system, and that is equivalent to saying that it gains entrance to the general circulation.

GENERAL SYMPTOMATOLOGY. All cases are common in displaying to some extent Disturbances of Sensation, Paralysis, Incoördination.

Sensory Changes are first in time of appearance, and also the last to go away so far as ordinary investigation or the patient's observation informs us, but Papoff asserts that faradic reaction is the earliest symptom, and Church says that a diminished knee-jerk has preceded by weeks the apparent onset of the disease. The history of a case will generally reveal that the patient began to notice sensations of numbness, creeping, crawling, stinging, burning, chilliness or heat in the extremities (paresthesias). They will be confined to the extremities for a few weeks, and then extend to the forearms and legs. They are not in stripes, or irregular areas, as in cases of simple isolated neuritis, but cover the extremities like a glove or stocking, reminding one of hysteria. They are not found upon the trunk, and literature furnishes only one case of an invasion of the face. This is the stage of nerve irritation.

Next we find that the nerve-trunks have become swollen and tender to touch, the muscles and joints as well, and there are spontaneous pains and cramps. These latter are most pronounced in the lower limbs. This is the stage of activity in the process of nerve-degeneration which has secondarily set up an interstitial inflammation in the connective tissue of the nerve-trunks, and also some ill-defined changes in the muscular elements.

Next is a loss of all tactile perception, and often for all varieties of cutaneous sensation, heat and cold, and pain, as well as ordinary touch. This is the stage of completed degeneration. An element in this loss, at times, is a diminution or abolition of the joint and muscle sense, originating according to Batten in a degeneration of the muscle-spindles, from which run fibres in the sensory portion of spinal nerves to arborize about the cells of Clarke's column in the cord, whence new axons go up in the direct cerebellar tract to the cerebellum. This mechanism presides over static equilibrium, and hence we may find total ataxia, with or without much paralysis. This is the pseudo- or neuro-tabes of the French, and often confuses this disease with tabes dorsalis, whose ataxic symptom is based upon a chronic form of this same degeneration.

Throughout all these changes, and final loss of sense perception, there runs an undercurrent of abnormal sensibility to pain, and therefore while the patient may fail to recognize the touch of a finger, or to wince at the prick of a pin, he may complain bitterly of the touch of a feather, or of lumps in the softest bed, or of knots in his clothing. It is anesthesia dolorosa, or painful anesthesia. This is most noticeable in poisoning by arsenic, and least in cases from lead, while alcohol presents most sensitiveness along the trunks of the nerves. Alcohol and grippe give rise to the most spontaneous pain. Pains about joints and in muscles are found in other diseases, and care may be needed to assign to them their true origin, but with care it will be found to be most pronounced along nerves which are distinctly swollen, and this with a symmetrical distribution is quite diagnostic. The area of these sensory changes conforms quite closely to that of the motor losses, and it varies quite considerably during the course of the disease. Convalescence is signalized by tingling and other sensations in parts that had become insensitive, and is usually perceived in the part last affected, and pains and over-sensitiveness may persist for a long time.

Motor Symptoms. The motor loss is usually rather relative than absolute, and would be of the grade termed a paresis rather than a paralysis. Rarely it may be sudden and very profound. The nerves typically affected in the upper extremities are the terminal branches of the musculo-spiral, which are the radial and the posterior interosseous supplying the following muscles: those of the posterior aspect of the forearm; extensor carpi radialis brevis, supinator (never in lead palsy), extensors (all the surrounding ones), branches to the carpal joint, these muscles are the extensors of the wrist and hand.

In the lower extremities the lesion is of the anterior tibial branch of the sciatic which supplies the following muscles: tibialis anticus, extensor longus digitorum, extensor proprius hallucis, peroneus tertius, and through the internal and external branches; the external to the extensor brevis digitorum and the tarsal joints. Internal unites with musculo-cutaneous nerves. The office of these muscles as a whole is to dorsally flex the foot, and to extend the toes. (See Fig. 60.) From these losses we get the characteristic wrist and foot-drop. These nerves are nearly, but not absolutely invariably the ones attacked, as in the arm we sometimes see, although in a very small percentage of the cases, the deltoid innervated by the circumflex. the biceps and long supinator, and the spinati affected first. The median nerve may be affected as well as the circumflex, and in such cases we find a distinctive deformity in "Claw-Hand." (See Fig. 57.) In the lower extremity the calf muscles, internal popliteal, and posterior tibial nerves may be paralyzed, and then the heel will strike first in walking, the foot will be inverted, and there will be dorsal flexion. Sometimes erratically the muscles of the eye may be affected, or the diaphragm may be paralyzed. In a marked case the foot-drop is complete, and while the patient is lying down the foot tends to the position of equino-varus. Milder cases are unable to dorsally flex the foot beyond a right angle. The toes are flexed and bunched, and can neither be extended nor separated. Muscular weakness can be estimated by their reaction to resistance. The gait is characteristic, as the thigh must be raised to flex the foot, the foot is then thrown forward by swinging the leg like a flail, the outer border of the foot is depressed, and the pendent toe strikes the ground. The outer side of the foot may be the first part to strike. The other foot is then swung around in the same way, giving a high knee-action, the "steppage gait" of Charcot. To continue the variations of gait, it may be stated that if the thigh muscles are paralyzed or greatly weakened, a possibility in alcoholic cases, walking may be impossible, or well-nigh impossible.

While distinctively a peripheral disease, it is not always so, but it is a possibility that every voluntary muscle of the body may be implicated, and the viscera even may be attacked. The trunk muscles are affected after the peripheral ones have been severely damaged. In such cases standing, walking, or even sitting, may be impossible. In very widespread cases we may find implication of the muscles of the eyes, face, and neck, of the tongue, throat, larynx, and diaphragm, of respiration, and of the heart. The patient may have to keep awake for long periods of time to make respiration possible. Such cases are most usually the effects of the toxins of diphtheria. The symmetrical character of the disease is especially well-marked in the muscular palsies, although it does not imply that the extent of such palsy will be the same in both limbs, for it is practically an axiom that the initial member is more deeply affected than its fellow which is attacked at a later date. The progress of the paralysis does not proceed according to any rule except this, and it is a very essential one to bear in mind, viz., the trunk is not affected until after the limbs. As an ordinary rule it is one forearm and then the other, and then one foot and then the other, or beginning in the feet and after affecting both, then, and then only, extending to the hands, but the rule is not by any means an invariable one. There may be a paralysis of a few muscles in one hand, and then a foot may be invaded, but however wide or narrowly circumscribed is the process, one is struck with the implications of a whole set of movements, which points to the nuclei in the cord or bulb as the actual seat of the damage, since combined muscular action, constituting movements, and not muscles, is represented in the spinal segments and cortex of the brain. Lead and diphtheria are most efficient in causing motor paralysis.

Reflexes. As a general statement it may be said, that from the outset the deep reflexes are greatly diminished (a primary exaggeration is possible, but rare), and the superficial are almost certainly so. Speaking exactly, it must be said that it all depends upon the degree of injury of the nerves, and the location of this injury. Just so long as any intact fibres remain in the nerve, there may be some reflex; and hence it may disappear in the knee, and be active in the wrist and elbow, but it is so nearly a universal truth that the extensors of both the upper and lower limbs are degenerated at the same time, that we may confidently look for a general reduction of all the deep reflexes to extinction, or nearly to that degree. The superficial are more universally absent, but Purves Stewart notes that in a case characterized by extreme hyperesthesia of the soles there was an exaggeration of the plantar reflex, and an exaggeration of tendon reflexes. The organic reflexes (bladder and rectum) are not impaired. Sometimes they will appear to be so in the last stages of very severe cases, but examination will show that it is only in cases where the mentality is so impaired that the calls of nature are not comprehended.

OPTIC NEURITIS. Occasionally it is associated with facial palsy. It is unusual in multiple neuritis, but it is occasionally found, and is of the retrobulbar type preceded by papillitis and choked disc.

Muscular Atrophy. This is a variable symptom, and yet the degeneration of the tissue of a muscle follows and keeps pace with its loss of power. It is localized in the same muscles, if present, as the paralysis. Fibrillation is present. There are cases where there is little atrophy, while there is great loss of power, and the reaction of degeneration is present. Voluntary muscles, and even viscera may be affected. Owing to muscular losses, a disuse atrophy may affect other portions of the body. Trophic conditions may be altered in any of their forms, as in vaso-motor control, irregularity of the secretions, both internal and external. The hair, and nails, and skin are apt to show changes, and there is general malnutrition. Sometimes it will be limited to glossy skin of the extremities, and an emaciation of the tips of the fingers, such as we see in wasting diseases.

Contractures. These are very apt to occur as a late symptom, and if present, will prove to be a great source of disablement, even if the power returns to the limb, and should therefore be antagonized at the earliest evidence of approach. They arise from four causes:

1. Lack of balance of the muscles on the two aspects of a limb.
2. In part from a natural predominance of the flexors.
3. In part from the action of gravitation.
4. In part from muscular fibrosis, and fibro-tendinous contraction.—(CHURCH).

From this condition arises anchylosis of the hands and feet, elbows and wrists, and even the hips and shoulders, and tenotomies may be needed to allow the heel to touch the floor.

Tremor. From the lack of muscular balance, and a general loss of muscular strength, we are apt to get all grades of tremor, to which symptom the loss of joint and muscle sense is also contributory, and they also produce the ataxia and incoördination which have already been referred to. It may show itself only as a clumsiness in movement. Almost always present, and may antedate the motor palsy (lead particularly), elicited only by attempts at motion, and most notable in arms, lips and tongue. This is marked in cases caused by alcohol. Especially evident in lips and tongue, and muscles of the face also. In lead it comes early, and may last as long as the paralysis; in cases from mercury, rarely from arsenic or opium, and may also come from tobacco.

Electrical Changes. At different stages of the same case we may find exaggeration in the outset to all qualities, and after a short time the reaction of degeneration, or there may be simply a reaction to galvanism, with a loss of response to faradism, which even in the severest cases is very rarely total. Erb mentions a case in which there was increased galvanic response; a myoidema lasting for several minutes. Osler quotes from Starr. Frequently faradic reaction alone is lost, but it may be a simple reduction of reaction to all qualities. It is a pathognomonic, that when the muscle alone reacts to galvanism it takes a strong current to produce any contraction, while in anterior poliomyelitis a weak current will cause a response for several months. Loss of faradic, and lowered response to galvanic, is a sign of multiple neuritis. Anodal closure contracture equal to, or more potent than cathodal closure contracture is the basic reaction of degeneration.

Mental Disturbances. A moderate amount of mental disturbance is very common in neuritis, and when slight it may be overlooked. It is most frequent among alcoholics; next in frequency come those caused by lead, and the infectious cases least of all. It may precede the onset, but generally developes along with the disability. This psychosis was first commented on by Starr, but in the year following, Korsakoff described it very fully, and alleged that it was peculiar to alcoholic cases. This has not been fully sustained, and the opinion held at present is, that upon a neuropathic heredity a confusional state might be developed by toxic substances, or by some acute infections. It is characterized initially by a change in the disposition of the individual, who shortly becomes a victim of insomnia. He then becomes subject to vague alarms, which at first possess him only by night. He may then become maniacal, and have hallucinations of sights and sounds, and of the special senses, after which he becomes depressed. The maniacal stage may be lacking. In any case there is a weakness of memory, and forgetfulness concerning, however, the near past alone. Under such conditions, misled by his hallucinations, he becomes utterly untrustworthy in his statements, and he will relate fictitious events with a wealth of detail. He shows a great lack of the power of concentration, and Oppenheim states, of orientation also. This is the power of correctly estimating his position in relation to the world surrounding him. Starr states that the depressive condition is peculiarly distressing, because his memory is too feeble to permit him to retain the consolatory admonitions of his friends and attendants, but it is to be diagnosed from typical melancholy by the fact that he does not indulge in self-recrimination as the author of his own misfortunes. Death may ensue from the gravity of the insomnia, or the violence of the cortical disease.

ALCOHOLIC NEURITIS. This is the most usual form, and is most constant in its symptomatology, and comprises more of the characteristics of multiple neuritis than that from any other cause. Lower extremities are most affected. Invasion is usually subacute. Pain is more pronounced than in any other toxic form and may be so severe as to cause insomnia, and thus pave the way for mental disturbance. Usually after an attack of gastritis, or exposure to damp and cold, the patient may develope a weakness in the lower limbs, which increases so rapidly that at the end of a week he may not be able to walk at all. Actual paralysis is less marked than in the others. Paresthesia is most intense and intolerable. The muscles are very sensitive. Cutaneous hyperesthesia is exquisitely developed in the palms, soles and tips of the fingers. The quadriceps extensor is attacked, as well as the extensors below the knees, and hence the high-action or "steppage gait" of Charcot is well marked. Atrophy is prominent, and fibro-tendinous contracture is pronounced. Ocular palsy is common. Vision is principally if not alone affected (amblyopia); it is bilateral, symmetrical, and insidious in approach, and must be searched for, as the patient may be ignorant of it. He may unconsciously have pronounced papillitis, retracted visual fields, color scotomata, and considerably reduced vision, and be ignorant of it. Squint is not common, but may be present; it may be due to an oculo-motor neuritis, and this is usual, and then the muscles are affected singly. It may, however, affect all at once. In this case it is due to nuclear involvement, and is usually accompanied by apoplectic features. Rarely dilatation, contraction and inequality of the pupils have been seen, but the pupillary reflex is not disturbed, and the Argyll-Robertson pupil has not been observed. Mental disturbance is most common in multiple neuritis from this cause, and has been discussed in the general symptomatology. Recovery is rapid under total abstinence from intoxicating liquors. Relapse is rapid and striking from the least indulgence. It may be without fever, or quite slight, or it may be pronounced. If a secondary rise of fever occurs it is often the sign of a pneumonia which is quite likely to be overlooked, as it is apt to be irregular in its localization. At this time the patient will be found to be tender to touch, and this tenderness will seem to be seated in the muscles rather than in the nerves, but if the trunk of the nerve be followed up, it will be found that it is the site of the greatest tenderness. The knee-jerks will be found to be diminished, but they will never be completely lost, under careful examination, since some fibres of the nerve are always preserved. The muscles of the trunk generally escape, and if attacked it will be a very late symptom (the reverse of what occurs in Landray's Paralysis). The affected muscles waste, and contractures occur, but they differ from the same symptoms occurring after a hemiplegia in that they are generally curable by care, and patient treatment. The muscles show the reaction of degeneration, and also a phenomenon which is diagnostic, viz., if a muscle be tested with an electric current, the resulting pain lasts for hours, while under ordinary conditions we should find it to be only a momentary distress. While the deep reflexes are variable, the skin reflexes are quickly diminished. Tremor is almost always present, and is widely distributed, but almost always spares the face. It is often only visible on attempts at muscular action, and is most evident in the arms, lips and tongue. Jactitation of the lips is insisted on by Osler.

Diphtheritic. It is clinically more common in adults, probably from the fact that its common manifestations as a pharyngeal, laryngeal, and pneumogastric paralysis are so rapidly fatal in an infant that death is hardly ascribed to the nerve-lesion. The throat and palatal muscles are most affected here, and at times exclusively, and as a result the patient may be embarrassed in swallowing, and the voice has a nasal quality, and food regurgitates through the nose. There are some cases which display a sensory and motor type of palsy like other forms of multiple neuritis, and this form is apt to be fatal, as it has an additional predilection for the nuclei of the medulla. The preceding illness is so recent, that it often guides us to the correct diagnosis, as the neuritis generally appears in from one to three weeks after the subsidence of the original malady, but may be present within a day or two of the appearance of the pseudo-membrane, or may occur in absence of this condition, but where the original disease has been so slight as to escape notice, the primary implication of the muscles of deglutition is a diagnostic point. The muscles of the soft palate and pharynx are the first to suffer, lips and cheeks may follow, the tongue also, and the legs next if it extends. Knee-jerks are often abolished before even the palate is invaded. The muscles of the neck are often so weakened that the head rolls about. The upper extremities are rarely invaded, wasting of the muscles is rare, contractures seldom develope, sensory damage consists of insensibility in the parts; severe pain and painful paresthesias are wanting. Ataxia to some degree is usual, Romberg's sign is seen, paralysis of accommodation occurs, so that there is loss of acuteness of vision for near objects, but there is no distinct pupillary paralysis. It comes at the time of the palatal paralysis. In children we may test by the use of a pinhole in a card, and in adults by print. In both cases the outline will be blurred. Church calls it paralysis, as he says that the neuritis cannot always be demonstrated. Hochhaus found only the muscles affected. Babinski paralyzed rabbits with the toxin from the Klebs-Loeffler bacillus, and found no changes whatever. In certain cases a peri-axial neuritis is plain. Why in some, and not in others, is not clear, but in all cases it is certain that the symptoms are due to toxins developed in the life history of the bacillus. The palsy may appear after any sort of a case, without regard to the location of the initial malady. It may follow from simple exposure which has given no evidence of its presence by sore throat or pseudo-membrane. Strabismus, double vision, and ptosis are not uncommon. Sexual impotence is frequently developed in males of adult years. Mental symptoms almost never occur. Diphtheritic paralysis is rapid in developement. Recovery is usual. If the palate alone is affected, recovery is usual in two or three weeks; if generalized it may last three months to a year. Rarely, paralysis is fleeting, and may be recurrent. Death is from inanition, cachexia, pneumogastric paralysis, inspirational pneumonia.

MERCURY. This is more an affection of the brain displayed upon the nerves than a primary affection of the nerves themselves. The motor and sensory palsies are present, but they are not so marked as the tremor, which is the prominent symptom. This tremor begins in the face, then goes to the trunk, and then into the arms and legs, and while at first it is only apparent on attempts at movement, it finally becomes constant. Lead may also show a like tremor, but it is in connection with colic, and the developement of a blue line on the gums, while with mercury we find a stomatitis, and a swelling and soreness of the gums, and a loosening of the teeth from a gingivitis.

ARSENIC. This is a very general form of paralysis with a mixture of motor and sensory symptoms. The finer muscles of the hand are more often affected than in the other forms, and it is most evident in the legs. Ataxia is prominent, and there is considerable atrophy. The cause can generally be found in arsenic taken as a poison; as a medicine (very rarely), or, at times, from gradual absorption from fabrics, or wall-coverings.

Osler doubts the effects of drugs as ordinarily administered, and is not sure that it can arise from the emanations from fabrics and wall-coverings, since the Styrian peasants have been known to take 8 grains of arsenious acid a day. It is as poisonous in a volatile as in a solid form, and if derived from fabrics it is from its transformation into a volatile form by the action of certain mould fungi. It has also been proven that dampness, and a temperature from 60 to 95 degrees Fahrenheit were sufficient to volatilize it. He had a case which developed a multiple neuritis from the effect of arsenic taken for the cure of Hodgkin's Disease. The patient took 4 ounces and 1 drachm of Fowler's Solution in 75 days, and during that time omitted 14 days, so that in fact he took 32.4 drops a day while actually taking the drug.

The resulting palsy is like that from lead, except that the legs are particularly affected, and in them the extensors and the peronei, so that the patient has "steppage gait." Early in the case, if one asks the patient to extend the wrist, and spread out the fingers, a tremor will be manifest. Reaction of degeneration may precede the palsy. From arsenic, symptoms like those of erythromelalgia have been seen. There is ridging of the nails. Marked tremor is rarer in arsenical poisoning than in any other, except that from opium. Dark mottling of the skin is diagnostic. In testing for the drug remember that the urine of healthy persons will show the reaction for arsenic in 30% of the cases.

LEAD. This intoxication usually arises from prolonged exposure to the metal through the use of liquids contaminated with lead from pipes, or containers, or cosmetics, or drugs, and is quite apt to be unsuspected, but may be accidentally developed into a neuritis by the administration of alkaloids. It is usually taken up by the mouth from hands soiled by the substance. Sources of lead may be hair dyes, face powders, styptics, injections which contain lead (all may delay in their action), fabrics, wall-papers, toys, medicinal preparations. A person may as ignorantly take lead into the system, as a person may alcohol in using a remedy said not to contain alcohol. It ought to be suspected in one who is engaged in an occupation in which lead is extensively handled, and even in the absence of such a history, if the neuritis has been preceded by obstinate constipation and abdominal colic. In chronic poisoning the muscles will show fatty degeneration, and fat in yellow patches. While the nerves show peripheral degeneration, the cord and nerve roots do not, and Osler does not say that any degeneration of the cells of the anterior horns has ever been found, but the hypothesis is that where there is primary degeneration it is due to such change.

Anemia marks all of these cases, and Cadwallader says that nucleation in the red blood corpuscles is present in even the slightest cases, and is diagnostic. The corpuscles are not reduced, however, in even the severe anemias, below 50%. Grawitz first, and then Pepper, White, and Vaughan, have found that it is diagnostic of lead intoxication to find with this moderate reduction of the red corpuscles a granular deposit in the cells, which is baso-philic to Jenner's stain and also to polychrome methylene blue.

The diagnostic blue-line is of two kinds. The first is between the gums and the teeth, and can easily be scrubbed off, while the other is a sulphide of lead deposited in the capillary loops, and with a magnifying glass can be seen to be in transverse lines, and not in a solid mass. It may be permanent or short-lived.

It is the most exclusively motor of all the forms of neuritis, and while there may be some pains in the arms, and some cramps, there are usually no other disturbances of sensation, unless the legs be affected, and then changes are below the knees. Pains in joints and legs are common at the outset, but there may be no sensory features at all. The upper extremities are almost exclusively affected, and certainly are the first parts to be attacked, and the hand most used will be the point of onset, but the paralysis will later become symmetrical. The site of invasion is the common extensor of the third and fourth fingers (the common extensor of the fingers is innervated by the posterior interosseous branch of the musculo-spiral), then of the wrist and then of the little finger. From this distribution we find wrist-drop to be the characteristic deformity, and if it extends upward, the supinator is commonly exempt, since the nerve especially affected is the radial branch of the musculo-spiral only. In severe cases we may find it extending to the muscles of the arm and the shoulder, later perhaps the legs, and in some cases the muscles of the trunk. Even when the trunk muscles have been affected, the prognosis is better than in the alcoholic type.

Fibrillary twitching ensues, and then prominent wasting. This may precede the palsy like a myopathy. Tremor is one of the commonest signs of lead intoxication. Retrocarpal tumor (Gubler's tumor) is frequent (no importance).

Madame Klumpke-Dejerine has divided the peripheral effects of lead into the following groups:

Ante-brachial type. Extensors of fingers and wrist involved, and therefore it is from the musculo-spiral. The supinator usually escapes. A swelling occasionally occurs on the wrist, which is known as Gubler's tumor.

Brachial type. Deltoid, biceps, brachialis anticus, supinator longus, pectorales rarely. This form is bilateral.

Aran-Duchenne type. Small muscles of the hand waste, also thenar and hypothenar eminences. Atrophy is marked. It is common in tailors, and may be the first manifestations of lead palsy.

Peroneal type. There are 100 cases of the arm type to 13 of the leg type. The lateral peroneal is affected, giving rise to palsy of the communis digitorum, and the extensor propris hallucis, and gives rise to "steppage gait."

Laryngeal form. This has been noted by Morell Mackenzie and others. It is a palsy of the adductors of the larynx.

Atrophy of the Scapulo-humeral type. This may follow the first form, or be primary, and is bilateral. It may be generalized, and begin in the wrists, and may involve every muscle of the arms below the neck.

Aphonia is common, as the laryngeal muscles are often attacked.

It sometimes sets up an optic neuritis, and sudden blindness may occur from this.

Mental disturbances may be like the general type of psychosis common to all the varieties of polyneuritis, or one peculiar to lead intoxication. This is an acute mania, which with the bodily symptoms reminds one of general paresis, but differs in the fact that expansive delusions are replaced by mental sluggishness. The common conditions caused by lead are delirium, convulsions, coma, hemiplegia, and all these in varying combination. The poison may induce arterio-sclerosis, and from this may arise lesions of the kidney, and this in turn may set up cerebral disturbances. This may be the cause of the mania.

DIABETIC. Neuralgias are apt to occur in the course of this disease, in consequence of the toxic condition of the blood, and also of the anemic condition. This engenders an enfeebled nutrition of the nerve, and the sciatic is the one most often involved. Multiple neuritis from this cause is exceedingly rare (Starr), but it is exceedingly interesting because it so closely resembles tabes dorsalis. Thirty per cent. of all diabetics lose their knee-jerk (von Ziemmsen, quoted by Starr), and they show ataxia and pains of all kinds, and also perforating ulcer. These phenomena seem to bear no relation to the amount, but to be dependent upon the mere presence of sugar, although with the reduction of the sugar the symptoms of neuritis pass away. Lack of an Argyll-Robertson pupil differentiates it from tabes.

CARBONIC ACID GAS NEURITIS produces peculiarly an anesthesia of the extremities, paralysis of the antero-lateral muscles of the legs, and later of the muscles of the upper extremities; perversion or increase of the tendon-reflexes; vasomotor or trophic changes; mental symptoms (particularly amnesia). Recovery follows in almost every case, but mental feebleness remains a very long time in even the most favorable cases (Gordon).

NEURO-TABES (Dejerine), Pseudo-tabes, is a form of multiple neuritis resulting from some recent intoxication, either alcohol, arsenic, or diphtheria, which is characterized by very rapid onset, by the presence of pain and ataxia, and almost no paralysis. Lobster poisoning has been known to cause it. It rarely affects the arms. It produces a rapid loss of the knee-jerk, and muscle-sense, and therefore Romberg's sign is present. This arises from a primary invasion of the muscle spindles. There is optic neuritis. The ataxia is a mixture of sensory and motor paralysis. Rarely there may be an excessive skin reflex, which is painful. Pain in general is common, and there is extreme muscular soreness. There is never rigidity of the pupils, nor is the bladder disturbed.

MALARIA. This has been known to cause multiple neuritis, but the outbreak has always been subsequent to an attack of malarial fever, or has shown its character by being promptly suppressed by quinine. It is peculiar in leaving the hands unaffected, and by attacking in the legs the anterior tibials, the flexors of the ankles, and the extensors of the toes. There is hyperesthesia

RECURRENT MULTIPLE NEURITIS. Sherwood, Ross, Dreschfield, Targlowa, Klumpke and Osler have described such a type, and Thomas has described a case. It appears that certain persons are susceptible to repeated attacks from various poisons, especially lead and alcohol, and this without renewed infection from the poison. Whether this is due to an original supersusceptibility, or to the prolonged effects of the original toxemia is not known.

BERI-BERI, OR KAKKE. A polyneuritis from a specific infection, occurring endemically in Japan, and epidemically in other tropical countries. No organism out of the many found has been agreed upon, but Wright, and Okita, and Kokubo have isolated specific microörganisms which when ingested elaborate in the duodenum a toxin, which upon absorption produces the disease. It has most constantly been ascribed to a diet of rice and fish, but accurate investigation has shown that a diet of *polished* rice (i. e., where the pericarp or inner hull has been removed), is certainly a constant factor in its production. "In the removal a saprophyte containing 'arsin' is freed, and mixes with the aleurone or proteid of the rice, whereas if the pericarp is left on, the arsin is encapsulated and harmless" (Starr). On the other hand Tahaki, the ex-surgeon-general of the Japanese navy, concluded, after ten years of investigation, that it was a disease of faulty nutrition, and depended upon a lack of nitrogenous foods in proportion to the carbohydrates, and he practically wiped out this disease by modifying the food. It is not contagious, but can be carried by persons. It certainly arises only when people are closely aggregated, as in institutions, and in tropical countries where these conditions are habitual.

SYMPTOMS. These are paralytic and atrophic. The lower limbs particularly, and those of the antero-external leg muscles, giving a characteristic gait. The phrenic and pneumogastric nerves suffer early, giving rise to respiratory and cardiac symptoms. The face and tongue are frequently affected; sensory disorders are mainly anesthesia and severe lightning pains. Edema of lower extremities is often great, and may flood the body cavity and invade its structures. Knee-jerk is lost early; right cardiac dilatation is frequent, with rapid heart action, and murmurs over the base. Mental disturbance like that of alcohol is frequent. (Absolutely rare, some say, and when present is hallucinatory—confusional in form, and of short duration.) All grades of severity from a simple weakness of the lower extremities with cardiac palpitation, to a pernicious acute form like an acute ascending myelitis, causing death in a few days. Intermediate varieties are always long in recovery, with a constant liability to death from pneumogastric accidents. A large proportion of cases is said to show the presence of intestinal parasites. Diagnosis is from its clinical peculiarities, and its manifest etiology. Prognosis in sporadic cases is good, while in epidemics fatality may rise to 60%.

Treatment is like that of polyneuritis in general, modified to some extent by the great liability to implication of the heart.

LEPROUS NEURITIS. The leprous bacillus can set up a neuritis. This causes a fibroid proliferation in the nerve-trunks, which often become nodular, and at length the bacilli disappear. The spinal cord is also invaded, and presents central cavities, especially in the posterior horns, and gray commissure. Prominent sensory symptom is anesthesia in discrete plaques, favoring in location hands, feet, forearms, legs and face. By spreading, large portions of the body may become anesthetic, and the deeper parts also, muscles, bones, etc. Muscular atrophies are comparatively slight, affecting mostly the small muscles of the hands, feet, and face. Atrophic conditions of the extremities may lead to mutilation of them. Evolution may be very slow, lasting years. Dissociation of cutaneous sensation may be encountered, like a syringomyelia of the Morvan type, probably due to the invasion of the cord previously noted. This variety is diagnosed from its clinical features, and from the discovery of the characteristic microörganism.

AUTO-TOXIC CASES. The great class of cases arising from intestinal fermentation and the infections must be diagnosed from their history. A well-marked case presents little difficulty in its recognition, since we find a symmetrical loss of power in the muscles situated first, and generally exclusively, in the extremities.

COURSE AND PROGNOSIS. In predicting the course of events it is pretty safe to assume that the disease will steadily grow worse for about six weeks, and by that time it will attain its maximum intensity, and wideness of distribution, then it will remain practically unaffected by treatment for from three to six months, and that recovery will take place in from six months to two years from the inception of the attack. Some cases are very acute, and rapidly invade the diaphragm and bulbar nerves, and are fatal in from six to twenty days. The probable duration of the attack may be predicted in a general way from the degree of its intensity, the wideness of its distribution, and the possibility of removing the cause. Recovery is the rule, even if cases have lasted a year. Diphtheritic cases are the most dangerous in the early stages, since respiration and cardiac action are more likely to be compromised, but in comparison with all other causes they are most sure of recovery. Lead, of itself, rarely is a cause of death, but since the patient's general condition has been previously deteriorated by the poison, the disease tends to be slow and disabling. Alcohol tends to be more fatal, since the patients are apt to have been previously debilitated by the poison. It is characterized by a great deal of pain, and that tends to exhaustion; patients are peculiarly liable to pneumonia, and it is often overlooked, since it is apt to have an unusual localization. Cases arising from auto-intoxication are the result of a very long-standing bowel disease, and so tend to be of long duration, since the exciting cause is still operative. If the invasion is acute and the localization wide, the patient may die in the early stages, but, if tided over this time, is apt to make a speedy recovery; but if the onset is slow it presages a chronic course. If the sphincters become impaired, it denotes an involvement of the cord, and whatever dangers that may imply. In any case, severity betokens initial danger and prolonged convalescence, while mildness presages rapid recovery. Good general condition insures prompt recovery. Contractures are generally curable. Tenderness in the muscles may remain long after muscular power has been regained, and so delay recovery for a long time.

DIAGNOSIS. The symptoms of all kinds are symmetrical in distribution. The deep reflexes are depressed while the sphincters escape. The superficial reflexes are abolished. Sensation is always perverted; at first paresthesia and later areas of anesthesia, while often there is some degree of spontaneous pain. The nerve-trunks are tender, there are tremor and atrophy; contractures are common. There is the reaction of degeneration, and mental disturbances are not uncommon. There is the presence, or history, of some competent cause.

ETIOLOGICAL DIAGNOSIS. Gowers says that in chronic metallic poisonings the arms suffer before the legs. Where the arms and legs are affected at the same time, it is extremely improbable that the neuritis is due to a metallic poison. Stewart (in Allchin) says that when from lead, the left hand is affected before the right only in left-handed persons. We get Gubler's tumor. An initial invasion of the proximal portions of the arms and legs, at the same time as the distal portions, denotes that the cause is not alcohol, but is probably cold, or a toxemia. If a few drops of alcoholic urine are added to a solution of Potassium bichromate 1 part, and Acid Sulph. fortior 300 parts, a bright green color will result. From arsenic the affection of the arms is soon followed by that of the legs, extreme sensitiveness, and bronzing of the skin, with cutaneous affections and ulceration of the gums, while from alcohol the legs suffer first. Cold or toxemia should be suspected where the muscles of the trunk or the bulbar nerves are affected early, and if there are grave constitutional symptoms. Diabetes should be suspected when there are irregular neuralgic pains, beside those located as we should expect in a multiple neuritis. In diphtheritic cases the knee-jerks are commonly lost, even if there is no paralysis of the legs. The associated symptoms should be carefully observed for diagnostic purposes relating to etiology. No form or aggregation of symptoms is, however, sufficient to certainly guide us to the cause of the attack.

DIFFERENTIAL DIAGNOSIS. No disease is more often confused with Multiple neuritis than Tabes. This is not to be marveled at, when we remember that both diseases have symptoms in common, and many *apparent* similarities. Knee-jerk is always lost in tabes, while in multiple neuritis it is more likely to be diminished. The Argyll-Robertson pupil is always found in tabes, but never in multiple neuritis, although the reaction to both light and accommodation may be reduced, on account of the inflammation of the optic nerve. Optic atrophy is common in tabes, and very rare in multiple neuritis, except in the form arising from wood alcohol. The tabetic is ataxic, while the sufferer from neuritis is paralytic; therefore while the tabetic slaps his foot upon the floor from lack of dexterity, the neuritic imitates this to some extent, since he has to elevate his knee to an exaggerated height in order to raise the dragging toe from the ground. The spontaneous pains and crises of tabes are absent from neuritis, where after quite severe pain initially in the affected nerve areas we find tenderness of nerve-trunk (never found in tabes), and of the muscles, and the tissues about the joints. The sphincters are commonly affected in tabes, and (except transiently, or apparently from mental changes) never in neuritis. Mental change is late in tabes, and is a dementia, while if present in neuritis it is hallucinatory or confusional in type. Neuro-tabes may be very confusing when both are moderate in degree, if lumbar puncture is impossible, and early recovery may alone differentiate it from tabes; but if the loss of muscular strength is slight, and the ataxia is so great that the patient cannot stand alone the disease is tabes. If examination of the cerebro-spinal fluid reveals lymphocytosis, the disease is tabes, and nothing else. If for any reason we cannot utilize this method of diagnosis a careful consideration of the symptomatology will keep us from error. Diagnosis between tabes and pseudo-tabes depends upon the following: onset more acute; some infection of toxic cause can be discovered; absence of bladder disturbance; absence of pupillary rigidity (seen only once in an alcoholic by Oppenheim, once by Eperon).

Acute Anterior Poliomyelitis usually attacks children who have previously been well, while polyneuritis is a disease of adults, except in those rare cases where children succumb to the intoxication of arsenic, diphtheria, malaria, or very rarely to alcohol. The onset is abrupt, and while it may be ushered in with fever, it is neither prolonged nor recurrent. There has been some previous cachexia or possible intoxication, the onset is insidious or sub-acute, fever is often prolonged, and may be recurrent. The resulting paralysis is immediately apparent; it is at first a general paraplegia or hemiplegia, or monoplegia, while in polyneuritis the paralysis takes days to develope. It is initially of definite groups of muscles, while this selective paralysis is a later feature of poliomyelitis, and above all, it is symmetrical in its distribution. Poliomyelitis may display some pains initially, but they usually are moderate in amount, and short in duration, while the varied sensory symptoms of polyneuritis are severe, and continue throughout the attack. In poliomyelitis the cranial nerves are rarely affected, and there are no mental symptoms, while both are fairly common in polyneuritis. Electrical changes are found in both, but while in polyneuritis the galvanic reaction fails soon after the faradic, in poliomyelitis it will persist, with much reduction, for some months. Finally, poliomyelitis always has atrophy following paralysis, and leaves permanent deformity, while in polyneuritis the atrophy precedes the paralysis, and lasting deformity is rare.

Cervical Pachymeningitis. Multiple neuritis affecting the upper extremities much more profoundly than the lower, might lead one to overlook the fact that they are implicated, and the question might arise whether or not it was a case of cervical pachymeningitis. The possibility of this disease might cloud the diagnosis, since it attacks the nerve-roots, and may produce a paralysis, wasting, and anesthesia affecting the upper limbs and the trunk, but not the lower limbs. There is no tenderness of the nerve-trunks, and there is distinct evidence of disease of the spinal cord, shown by root-pains in definite areas. Examine the spinal fluid for lymphocytes, as it is usually syphilitic.

Myelitis is a most commonly confused condition. When this disease affects the posterior structures of the cord, so as to give rise to ataxic symptoms, it reminds one of neuro-tabes, or pseudo-tabes, but there are always some symptoms which can come only from some lesion of the cord itself. Complete loss of power in a day or two is strong presumptive evidence of myelitis. The paralysis extends from legs to trunk, and perhaps next to arms. In contrast to multiple neuritis, the pain of myelitis is in the spine, and the paralysis is of the legs (a paraplegia), or of the legs and trunk, and there is rigidity of the muscles, while in multiple neuritis there is flaccidity. There is no electrical reaction of degeneration, except in those muscles innervated from the segments actually implicated in the disease. The deep reflexes are exalted, unless their centers are destroyed, and then they are absolutely lost. The muscles of the back are always paralyzed to some extent, and this loss is in the form of a girdle. The sphincters are always affected, and the atrophic process is so localized, and so acute, that bedsores are common. There is heat in the spine, and if there are anesthesias and paresthesias, they occur in areas like stripes and girdles, and are on the trunk, and affect the upper parts of the limbs before the lower.

In multiple neuritis there is no pain in the spine or trunk, except in a general extension, late, in a very severe case. The paralysis tends to be in some degree simultaneous in the four extremities; never from arms to legs, or legs to arms with an intermediate involvement of the trunk as in myelitis. The muscles quickly show the reaction of degeneration, the deep reflexes are very rarely exaggerated, and then only at the outset, but tend to fade quickly to complete, or almost complete extinction. The muscles of the trunk suffer only in the latter stages of the most extreme cases, and never in a girdle-like distribution. The sphincters are never permanently involved, and the trophic changes are never severe enough to produce bedsores. The tenderness is along nerve-trunks, and in muscles, and about joints, and never extends above the knees.

Landray's Paralysis has been said to present a confusing picture, but the diseases have few points of actual similarity. It has no sensory symptoms, except occasionally some initial numbness and tingling, which is in strong contrast to the severe pains and the exquisite tenderness of the nerves in multiple neuritis. (A case of my own started with tingling in lips, and weakness in legs.) Both have paralysis, but while that of Landray begins in the legs, and moves steadily upward, involving next the trunk, then the arms, and lastly the face, that of multiple neuritis may begin either in the arms, or the legs, and goes to the other extremities, never affecting the trunk, except in those rare and severe cases which are so marked as to permit of no error in diagnosis. The sphincters are rulably exempt in both diseases, but while this rule is occasionally infringed by Landray, it is never permanently by multiple neuritis. The course and termination also are strikingly dissimilar. Landray paralyzes the whole muscular system in a few days, and with few exceptions is fatal in from three days to two weeks, by paralyzing respiration and cardiac action, while multiple neuritis consumes from ten days to two months to produce total disability in the parts affected, and tends almost certainly to recovery. Fever to about the same degree may accompany both diseases. The only exception to the gradual onset is in those very severe cases of alcoholic intoxication where the legs may be totally disabled in a few hours to a day or two, but even here the distribution still conforms to the rule as stated. for the trunk is either not involved at all, or not until a much later period.

Hysteria may co-exist with multiple neuritis, and these diseases have been confused when in pure type, although there are essential and ascertainable points of difference. Both present paralysis, anesthesias, hyperesthesias, atrophies, and changed reflexes, but with a difference. The paralysis of hysteria is of a section of the body as thought of in the mind, as of a leg, or both legs; or of an arm, or of one side of the body, or of the whole body; whereas the paralysis of multiple neuritis is of such groups of muscles as are innervated by certain specific nerves. Moreover the paralysis of hysteria has never been a loss of the power of extension, while that of multiple neuritis is peculiarly and distinctively of this character. The anesthesias of hysteria are bounded by straight lines, at right angles to one another, and sharply defined at the borders, and such areas are scattered about the body practically without exception, having at the same time some such areas upon the trunk. There is always some anesthesia of the special senses. In multiple neuritis on the contrary, the anesthesias avoid the trunk, while on the limbs the areas follow the distribution of certain nerves, and the borders are very poorly defined, since the sensory nerves of the skin infringe to some extent upon the borders of each other's territory. In the general distribution of these anesthesias upon the extremities, both are glove or stocking-like. While the hysterical patient complains bitterly of hypersensitive areas, the calm expression of the face during pressure upon such areas, if the patient be off his guard, negatives the truth of his statement, nor are the nerve-trunks especially tender, nor are they ever swollen. The atrophies of hysteria are slight, and result only from disuse of the part, and never show the reaction of degeneration, while those of multiple neuritis are the contrary in every particular. Hysteria shows no atrophic changes in the skin, while they are frequent in multiple neuritis, the peculiar skin changes of hysteria being insensitive areas which do not bleed upon puncture. These, together with the narrowing of the fields of vision, and a reversal of the area in which various colors can be perceived, constitute what are known as the *Stigmata* of hysteria, and are not duplicated by the symptoms of multiple neuritis. The deep reflexes are exaggerated in hysteria, and after a primary exaggeration, which is not usual, are diminished almost or clear to extinction in multiple neuritis. Hysteria attacks young people, mostly women, who have been exposed to some emotional strain, and are of a neuropathic constitution, while multiple neuritis affects persons who are somewhat older, and who have been exposed to some intoxication, or who are the victims of some cachexia.

Neuralgia can be diagnosed from multiple neuritis from the fact that it presents more spontaneous pain, and also that this pain is liable to exacerbations at certain fixed times of the twenty-four hours. It is almost always unilateral, as opposed to the symmetrical distribution of multiple neuritis, and while there will be areas of exquisite sensitiveness for a short distance along the nerve-trunks, this varies greatly during different periods of the same day, while that of multiple neuritis is of the same grade, while it continues as a symptom, and extends along the whole nerve, or for long stretches. Neuralgia, also, after a time, ceases to confine itself to the nerve or nerves originally implicated. The nerves in neuralgia do not develope the reaction of degeneration. It must be remembered that multiple neuritis may sometimes appear initially as a neuritis of a single nerve. There is no swelling of the nerve in neuralgia.

Spinal Meningitis. The onset is abrupt; the principal feature is pain, which is always girdle-like, but not to the exclusion of shooting pains, and those radiating down the limbs, since they follow the distribution of nerves, and may go up to the head and neck also. These are the so-called "Root-pains." On account of the same irritation of the roots where they emerge from the cord, we get cramps in muscles, twitching of limbs, opisthotonos (a rigid backward curvature of the spinal column), and while the deep reflexes are at first exaggerated, in the end they are lost. The sphincters are involved. There are all varieties of disturbance of superficial sensation. There is some degree of paralysis, "tache cerebrale," and Kernig's sign.

Spinal Hemorrhage. The manner of onset of its rapidity, and the distribution of the subsequent pains are quite diagnostic. In any case the onset is abrupt, and often the patient will suddenly fall with a sense of shock of some kind in the back, and immediately find himself paralyzed in his lower limbs. This is the more impressive and confusing to the sufferer, since his consciousness, and acuteness of intellect remain unimpaired. It is soon marked by pain in the back, which will always show some girdle-like distribution, and in addition will radiate over other parts of the body. The sphincters are involved, and the deep reflexes will be changed, either increased, or lost, according to the site of the lesion. The cranial nerves are spared.

TREATMENT. Removal of the cause is the first measure to be thought of: removing a person from a poisonous occupation, or preventing the absorption of more of the poison from the water which they drink, or the air which they breathe. It has been found that even old drunkards bear abstinence well under these circumstances, if they are well-fed, and kept warm. Lead has been diagnostically stated to be easily eliminated by the administration of Iodide of Potash. In this procedure, however, it has been asserted that there was a great danger of flooding the body with a soluble iodide, by which a more severe and widespread poisoning was produced. In view of this, T. A. W. Ogg advises that the use of Iodide of Potassium should be accompanied by the free drinking of Sulphur water, by which the soluble iodide of lead is changed into an insoluble sulphide, and so passes harmlessly out of the system. Professor Dixon Mann, however, states that elimination is a very gradual process, uninfluenced by any of these means, but is slightly accelerated by warm baths. There is no particular antidote for Mercury. Antitoxic serum in diphtheritic cases. Comby reports a child of four years cured in three days of a severe paralysis. An old case, which had resisted other treatment, in a short time responded. He thinks all cases should get it, as the virus may not have exhausted its virulence. Others deny its efficiency, and consider it absolutely bad.

In the acute stage, rest and warmth for the affected parts is about all that is efficient locally, and this is best assured by keeping the patient in bed, with the affected portions of the body swathed in cotton, and wrapped with a flannel roller; but when severe pains appear, warm fomentations may be applied for half an hour, and then replaced by the compresses for four hours. If a case is very severe, a water bed may be needed on account of the tenderness of the muscles. It not only abates the pain, but supports the patient if he is very weak, and also lessens the danger from a debilitated heart, and impaired respiratory muscles. From an early date, a daily warm bath and gentle friction allay pain, and reduce the amount of muscular degeneration.

Alternate hot and cold sponging have been advised by Mills. Immersion in a very hot bath for twenty minutes to a half hour will quiet the pains in some cases. If the patient's heart is not weak, vapor or Turkish baths are valuable. Fresh air and sunlight are essential aids to recovery. If the muscles of deglutition are invaded, feeding should be by nasal or stomach tube, or rectally if necessary, since ample nutrition by some route is an absolute essential, and it should be simple, nourishing, and sufficient in quantity.

The bed clothing should be supported above the patient by a cradle if there is the slightest tendency to foot-drop, and the feet and legs should be placed in normal positions, and kept there by pillows, sand-bags, or removable splints, so as not to forego the daily bath and friction. Passive motion of all joints in full extension should be practiced daily, for contractures tend to occur, but these are generally unnecessary if the limbs are strapped or put upon splints at the first sign of deformity, and are then exercised. Even while the pain is present mild galvanic brushing may be beneficial, but as soon as repair begins, the muscles must be daily exercised by means of the galvanic current, always using large electrodes, and avoiding fatigue. The first response will be obtained by means of static sparks. As soon as the faradic response appears, it should be the current used, and each muscle should be given two or three vigorous contractions daily. Swedish movements against resistance are useful. Atrophy and degeneration can best be antagonized by persistent massage. If, after the attack, the toes tend to bend under the foot, and so impede walking, it is best to put them in a corset-shoe, and build up the heels. A rubber muscle will aid in another way. The patient should not get up until pain and tenderness have subsided, and there is evidence that the process of repair has become established.

Electrical Treatment. When the case has failed to respond to such measures, and we find that the muscles are unresponsive, or have been neglected, and contractures and deformities are present, we must wake up the muscles, at first with static sparks, and then follow with galvanism. Contractures must be broken up, if necessary under an anesthetic, and fixed by a splint or dressing. Faradism is best for cutaneous anesthesias, and chronic wrist-drop should be corrected by apparatus.

Electricity cures rapidly in those cases where reactions have not changed to the point that faradic irritability is lost, and in these cases the faradic current is preferable. When faradic irritability is lost, galvanism may be used. When treating a case with the faradic current the high-tension coil should be used, and the amount of current should be small; not enough to produce contraction. The application of one pole should be made to the affected nerve, while the other is over that section of the spine which gives rise to the nerve. In using the galvanic current, treatment should be given labile with the anode over the affected nerves, and the muscles affected by them, and the cathode resting over that section of the spine which gives rise to them. The special point in the treatment of this disease is to use a very weak current, five to ten milliamperes, but continued a very long time, at least twenty-five or thirty minutes. This treatment has proved far more successful than the administration of stronger currents for a shorter space of time. Static electricity as a routine treatment does not give as beneficial results as galvanism and faradism, and, if employed, the static spray administered for some time, twenty to twenty-five minutes, gives the best results, and is most beneficial in those cases where sensory changes are present. The sinusoidal current is often valuable in stimulating regenerating nerves where the other modalities are not efficient, or where the static sparks are not available, or cannot be employed. Cohn recommends labile combined galvano-faradic treatment of the affected parts. Where irritability has ceased all electro-therapeutic measures are useless.

THERAPEUTICS. Our literature is full of remedies, but the most accurate reports seem to indicate that our results are just as dependent as in *physiological* medication upon finding and combating the cause. The most success comes from ascertaining the cause, and treating the patient as we would if the disease had different manifestations. If, therefore, the case is rheumatic, we should at once think of Bryonia, Rhus, Cimicifuga, or Apis. Symptoms might occasionally turn us to one or other of the more unusual drugs, but if we keep in mind not only the existence, but also the *comparative* value of symptoms, we shall be likely to keep pretty closely to the drugs mentioned. In alcoholic cases the usual prescription will be either Nux vomica, or Cimicifuga. Cases which develope marked tremor would seem to call for Argentum nit. conspicuously, among others, but it has not seemed so valuable as in some other diseases marked by this symptom.

Aconite. In the earliest stages, but never if tingling is not a marked symptom.

Argentum nit. Post-diphtheritic, marked by dilatation of the pupils.

Arsenicum. This is especially indicated in lead palsies. The pains relieved by this drug are worse at night, are better by heat, and there is marked mental restlessness.

Apis. Shows numbness with burning pain, and great sensitiveness.

Agaricus. The presence of much muscular jerking, and twitching.

Arnica. Where there is not so much pain as soreness, the patient shrinks from being even approached, and there is aggravation from motion.

Belladonna. When high fever marks the attack.

Bellis perennis θ, 5 drops in a half glass of water, a teaspoonful hourly has often produced perfect cure in alcoholic cases which exhibit an extreme sensitiveness of the nerves, and a bruised feeling similar to that found under Arnica (Butler).

Bovista. For the stage of numbness and tingling.

Cocculus. Tingling of limbs with paralytic trembling.

Colocynth. In infrequent cases.

Calcarea carb. For the chronic conditions.

Carboneum sul. In same. Especially suited for cases affecting the territory of the median and radial nerves. Pains and jerkings, particularly at night.

Ferrum phos. Dead feelings in the hands, and a soreness of the muscles upon motion.

Gelsemium. Paralysis with marked tremor. Markedly motor.

Hypericum. Is characterized by as great fear of approach as Arnica, but it has more sensitiveness, and the spine is especially sore to touch. It is more often a neuralgic remedy than one for neuritis.

Lathyrus, and Ustilago maidis. Have produced the lesions in nerves, but the clinical results have been disappointing.

Magnesium phos. Numbness of hands, with lightning pains running up the limbs.

Nitric acid. Is called for by a lancinating pain, but it is more often neuralgic.

Silicea. Has good clinical reports.

Zinc. Is highly spoken of, but has never seemed to favorably affect an organic condition.

In the stage of fever the physiological treatment has been antipyretics of all kinds. In alcoholic cases Citrate of Potash, Nitrous Ether, Tincture of Cinchona, and Digitalis if the heart is weak. In rheumatic cases the Salicylate of Soda, but if the patient be also alcoholic the Salicylate of Potash should be substituted to avoid the combination with, and retention of, the uric acid by the Soda. Asparin is now preferred to either. No drug is specific, and while Mercury is more efficient in the interstitial varieties of neuritis, it still should be tried in polyneuritis when the nerve-sheaths seem to have become affected. Antipyrine and Mercury in small and repeated doses are of great value in rheumatic cases. Mills advises Gaultheria for rheumatic cases. In toxemias like septicemia give Tr. Perchloride of Iron in doses of 20 to 30 drops t.i.d. Mercury and Kali Iodide for syphilitic cases. After the first shock is over, Iron, and Strychnine in small doses, and Quinine. Others say Strychnine in increasing doses.

Iodide of Potash is used extensively, but for some unknown reason is good only for the sensory forms. Arsenic, in small doses also, in the sensory cases only. It is advisable to add one-twelfth to one-quarter grain of Cocaine to whatever other remedy is given if the patient be an alcoholic, as it stops the craving, and renders the stomach more tolerant of food.

Quinine very freely for the malarial form. This form can only be differentiated at times by the action of this remedy.

The use of anodynes is always likely to become important, and the Coal-tar series is usually better than the opiates. Cocaine hypodermatically one-fourth of a grain seems to aid regeneration, and does not simply curb the pain. Sleep is most certainly produced by a mixture of Bromide and Chloral, but Trional, Sulphonal, Chloralamid, Phenalgin, and Antipyrine, are useful in about the order given.

Mental excitement is most surely antidoted by Hyoscine 1-400th to 1-150th of a grain.

After the febrile state use a general tonic treatment of Strychnia, Iron, Quinine; Arsenic in small doses, as it may increase the trouble. Codeine and Cocaine are better as anodynes than Opium, as the habit is less easily formed. In lead cases purge freely, and give Opium in the colic stage.

NERVE-INJURIES.

Nerves are frequently exposed to injury, which may range from a slight degree of compression, and of short duration, to the complete rupture of the whole nerve-trunk. An arm is placed in an uncomfortable position for a few moments, and we recognize by the resulting tingling (paresthesia) that the nerve has been compressed, or has been rendered ischemic by an interference with its nutrition. The force was slight, and its duration was short, but its symptoms differ in degree only from those following the most serious injury.

Etiology. Simple pressure for fifteen seconds, if severe, will break up the myelin, and set up a neuritis of some duration, but the nerve-fibre is not susceptible of injury in so short a time. Longer pressure is liable to injure the fibre, but this is not to be precisely gauged by time. All traumata of peripheral nerves act by disintegration of the myelin, or of the fibre itself, thus producing a solution of continuity in the nerve. Fractures or dislocations of bones frequently rupture nerves. They may be caught in callus, or in new connective-tissue about fractures, or in cicatrices. They may be pressed upon by aneurisms or tumors; it is possible to injure them by the too forcible contraction of a muscle, or they may be caught between bones thrown into unusual, or unusually prolonged apposition, as in some surgical positions during narcosis, or between the bones of the pelvis and the head of the child in gestation, or, more commonly, in parturition.

Pathology. Slight compression is often ischemic only, and results in only a temporary functional loss. Longer compression disintegrates the myelin, and seriously affects the nutrition of that section of the fibre for some time. Crushing disintegrates the whole nerve-trunk, while laceration tears it in two, and may separate the fragments to such a distance that reunion is impossible if unaided by surgical measures. If the traumatism has been so severe as to expose the ends of the torn nerve, primary infection may be added. In all, except the last instance, the resulting condition is not an inflammation, but is the Wallerian degeneration described in a previous section of this same article. The resulting degeneration in the

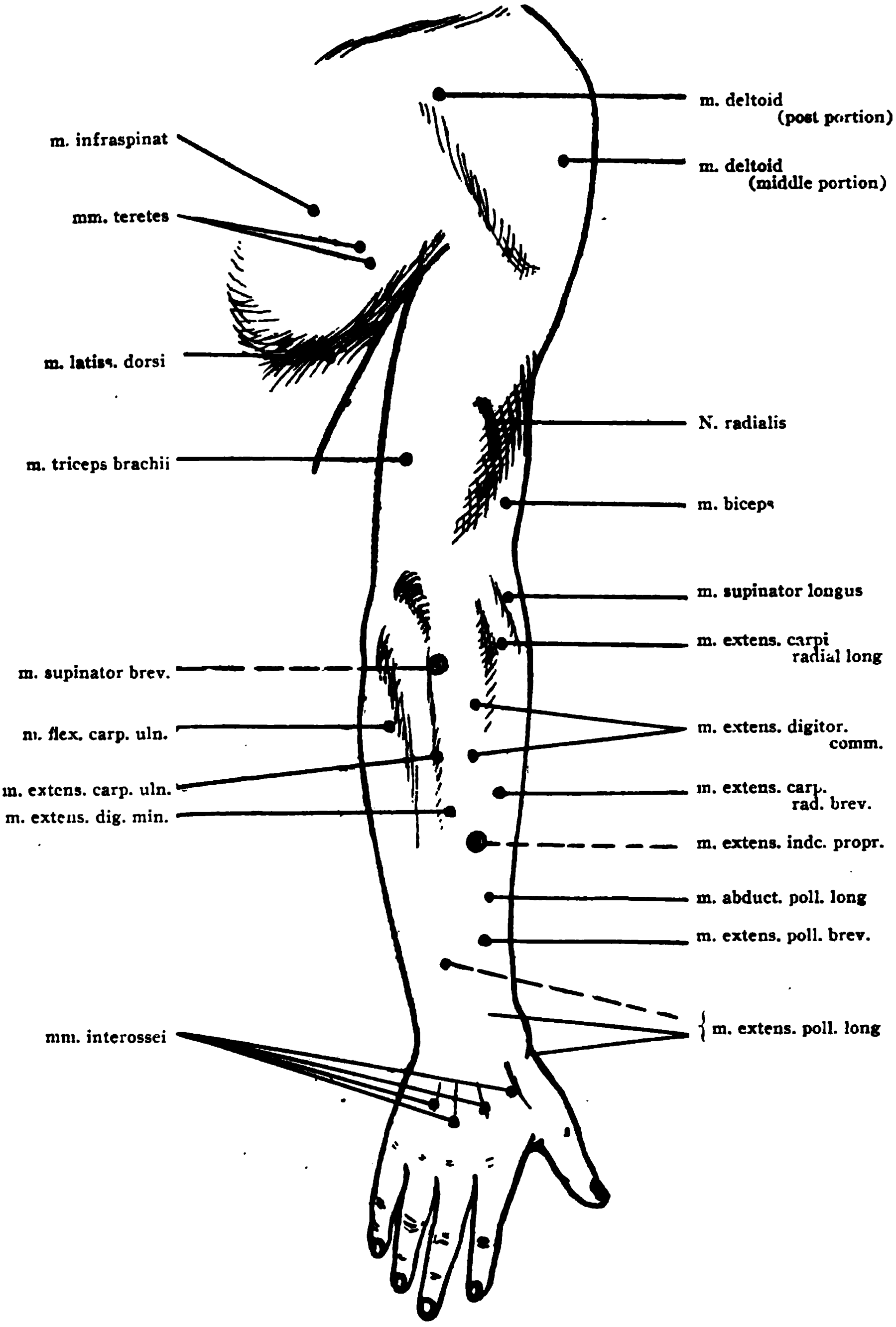


FIGURE 39. (From King.)

central stump does not extend definitely more than one-third of an inch, although some degenerated spots may be found clear back to the neuron cell, but peripherally it involves the whole length of the nerve, and also its terminals in the muscles and other structures which it innervates. There are possibly no definite trophic cells in the cord, but if the sensory stimuli from the tissues fail to be transmitted to the cells in the anterior part of the cord, atrophy always results.

Repair is inaugurated by the proliferation of fine fibrillæ from the central stump which seek an anastomosis with the fibres of the peripheral portion. They ramify the new connective-tissue in all directions, and when they finally are re-united, they develope a coating of myelin, and regenerate again into a perfect nerve-trunk. A true union of the ends of divided nerves rarely if ever takes place. The process of repair after physical reunion is not, as yet, understood.

SYMPTOMATOLOGY. In slight and temporary compression there will be numbness of the parts, and a degree of weakness in the member, which soon passes away spontaneously, or after a little rubbing. In severer cases of the same type the condition may last for some weeks, but, if the force has been sufficient to actually involve the integrity of the nerve, disintegration will ensue in from five to eight days. In such cases the resulting symptoms will differ only in degree in all forms and degrees of injury, except that where the lacerated ends of the nerves have been exposed to the air a greater degree of pain will ensue. In all accidents to mixed nerves we shall be struck by the relatively greater loss of motor than of sensory function. This depends upon two factors. Sensory fibres are more resistant to injury, and owing to anastomosis the sensory innervation of an area is not so dependent upon the integrity of a single nerve. The first symptom of such an injury is a change in sensation in the dependent part (paresthesia). It is at first an increase, followed later by all degrees of diminution and loss; tingling, burning, numbness followed by diminution of the appreciation of cold and pressure, then of warmth and pain. There is loss of the motor power, since the tonicity of muscles decreases after forty-eight hours; next there is flaccidity, abolition of the tendon-reflexes, and then muscular atrophy. Muscular twitchings and spasm (probably reflex) are not generally prominent, but there is at times fibrillation of muscles. Muscle and joint-sense are rarely disturbed. At first the sensory disturbances are not confined to the area of the nerve actually injured, but are quite widely distributed, yet after the initial shock has passed away these sympathetically disturbed areas become normal.

Trophic symptoms, aside from atrophy, are common, and the skin is affected by simple atrophy, and also by eruptive disturbances of all degrees, corresponding to the gravity of the injury. The joints and nails are also subject to disintegration. Most pronounced in septic cases.

Vaso-motor irregularities develope and the skin becomes blue, there is an increase, or, more rarely, an absence of sweat, and the surface may be cold from ischemia, or hot from congestion. Electrical changes, and some grade of the reaction of degeneration, or simple diminution of response are found.

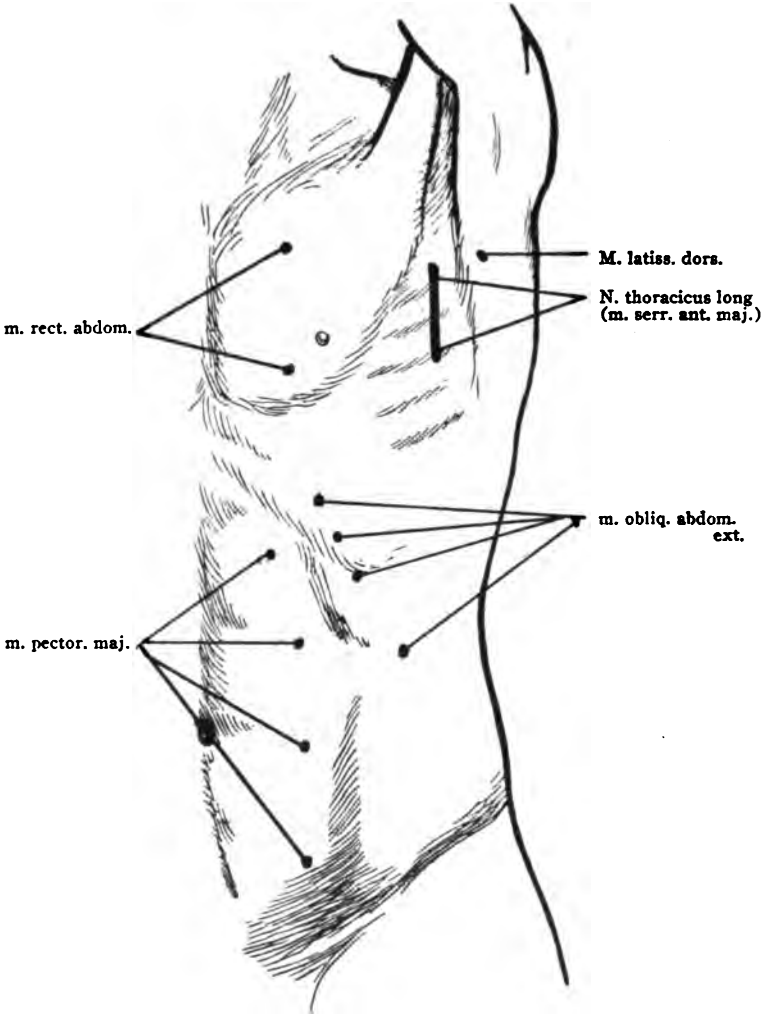


FIGURE 40. (From King.)

COURSE AND PROGNOSIS. Full degeneration takes place in about two weeks. Simple pressure paralysis may recover in a few weeks, or even days, but if severe, it may be a matter of many months, and if division of the nerve has occurred there will be regeneration only if the parts are still in comparatively close apposition. If a person suffers a relatively slight injury while under the influence of a narcotic, or chronic intoxication, or anesthetic, or the results of an infection, the damage will be much greater, and recovery more doubtful or delayed. Electrical reaction is the best guide. If the reaction of degeneration is present, recovery is doubtful, and the

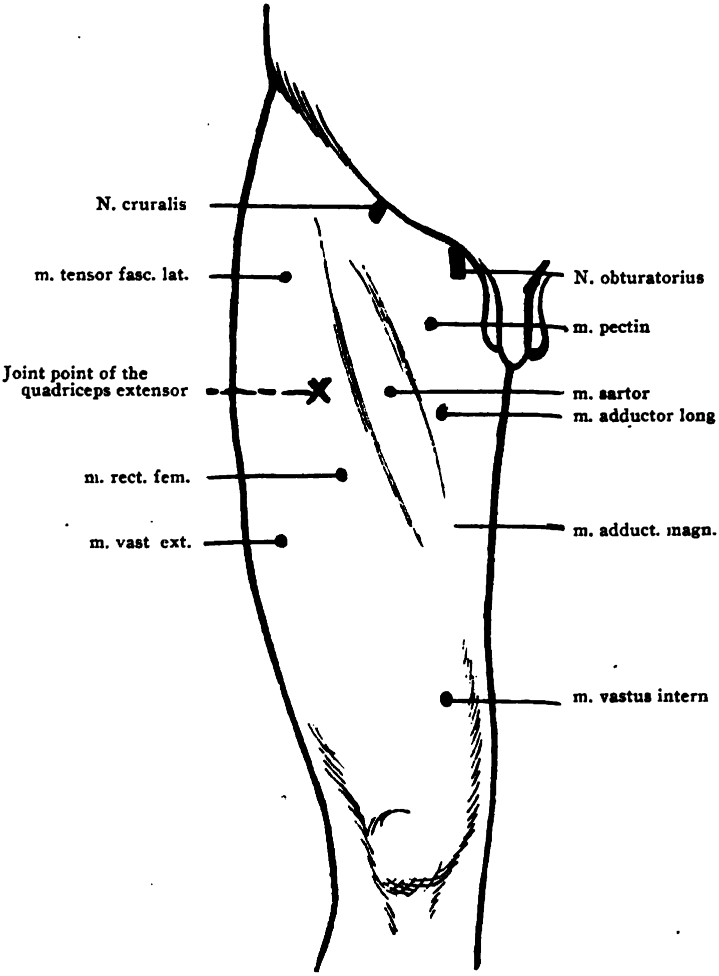


FIGURE 41. (From King.)

best that can be hoped is recovery in months; three or four are the least. If after the second week we find a normal response a speedy recovery may be predicted. A partial reaction of degeneration gives ground for a relatively good prognosis for recovery, but no time-limit can safely be set. Cases have recovered, even after twenty years, where removal of a physical obstruction to repair was possible.

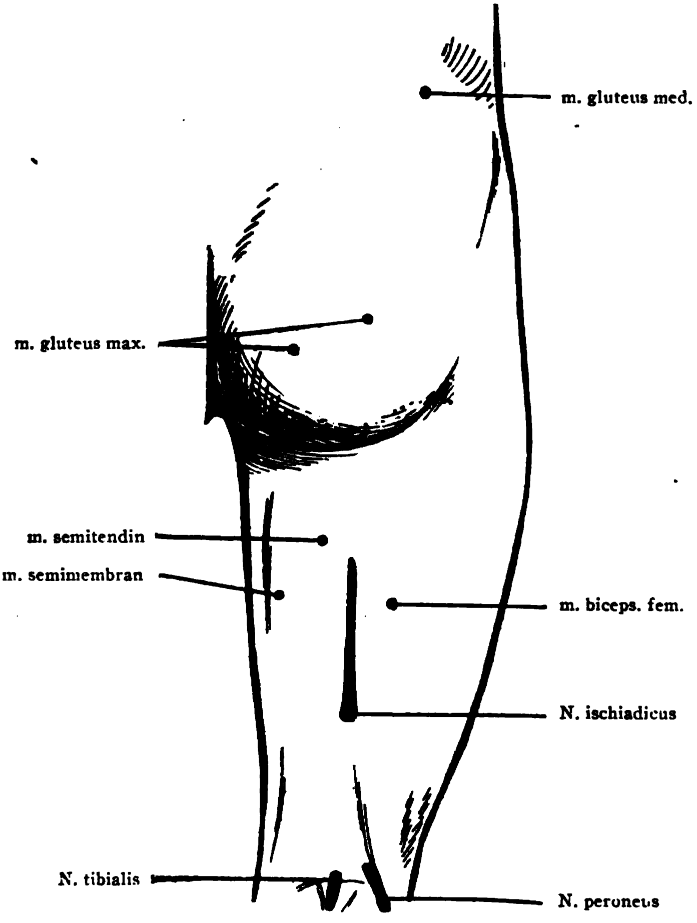


FIGURE 42. (From King.)

DIAGNOSIS. In traumatic neuritis the diagnosis lies between that, and a paralysis resulting from contusions and injuries to joints, and the effects of splints. In joint-contusions, while they may present the same paralysis and muscular atrophies, we find only quantitative changes to electrical stimulation, with intact sensibility. In the ischemic muscular contractures which follow the use of splints, we find a history that the hand has swelled, has been painful, and that there have been contractures in flexion in the hand and the fingers. The possibility of movement is lost, and it is difficult to passively move it, and it causes pain. The diagnostic points are the hardness of the muscles, the stiffness of the joints, the absence of any reaction of degeneration, and the severity of the sensory disturbances. It may be very difficult at times to decide in old cases whether or not contractures are due to a neuritis, or to peripheral injuries. Here the presence of scars may be the only guide.

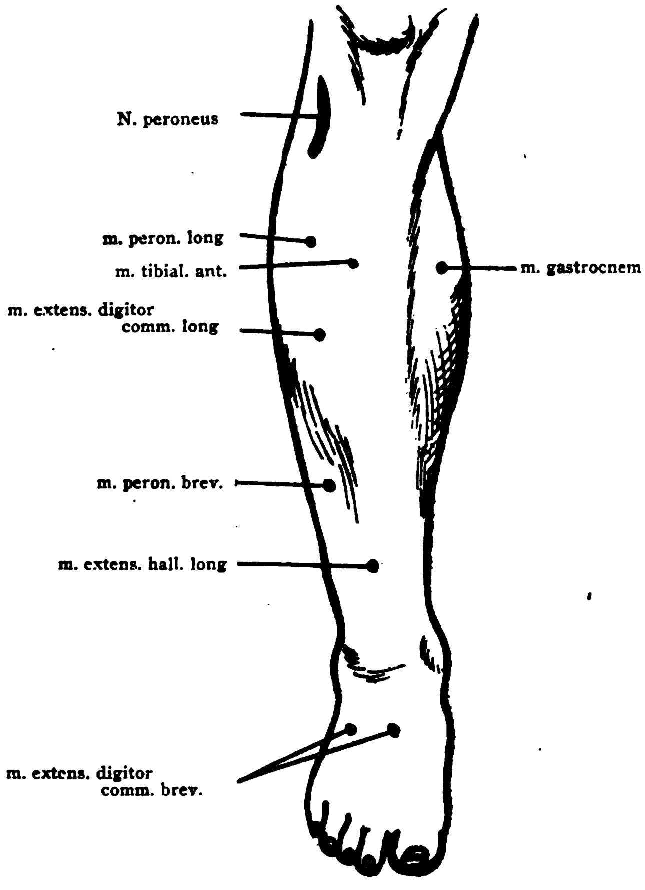


FIGURE 43. (From King.)

TREATMENT. This does not differ in many cases from that of ordinary forms of neuritis, but in others surgery may be the only treatment initially, to be followed by massage and electricity, as in the other form. Nerves must be relieved from pressure of tumors, exudates, or callus, and intervals between stumps must be bridged by decalcified bone, or strands of gut, or longitudinal nerve-suture must be performed.

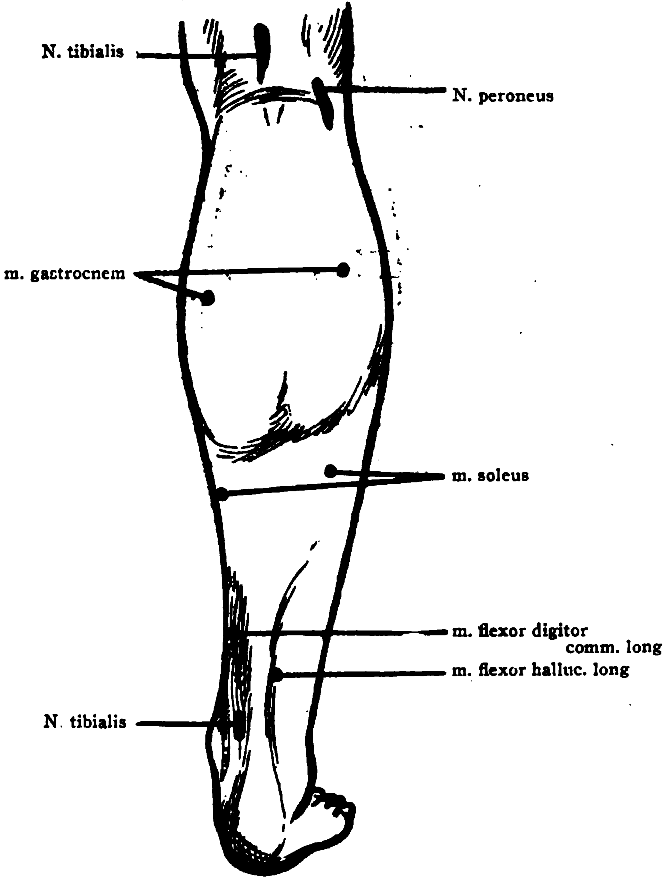


FIGURE 44. (From King.)

NEURALGIA.

DEFINITION. This is the term applied to a severe paroxysmal pain felt in the course or distribution of a nerve, or in both localities.

DIFFERENTIAL DIAGNOSIS. This is from Organic Nervous Disease, Neuritis, Hysteria, or Psychalgia, Meralgia paresthetica or Melalgia, Herpes Zoster, Referred Pains, Epicondylalgia (Remak).

AGE. It is most common in the active period of life when the organism is most exposed to rigorous conditions and severe strain, and the tendency to it decreases with advancing age, since conditions are apt to be less trying, and the terminals of the sensory nerves and the recipient cells are less susceptible to irritation.

SEX. More females than males are attacked.

ETIOLOGY. The various intoxicants are capable of inducing a neuralgia, and the products of auto-infections, whether they arise from malnutrition, or inanition, or from the cachexias. Malaria, syphilis and diabetes are all potent causes. The toxines of infections produce it very frequently; refrigeration also, especially if accompanied by dampness, electrical changes, and sometimes emotional strains. These must all be considered merely exciting causes, since at the root of all attacks of neuralgia except those arising from specific toxemias we shall find one of two predisposing causes, viz., a neuropathic heredity, or malnutrition. With the same degree of exposure or strain one person will develope a neuralgia, and the other will show no such disturbance, and the determining factor will be that one person comes of a neuropathic ancestry, while the other does not, or the one will be anemic from previous malnutrition, fundamental or acquired, while the immune person will be in vigorous health. The well-nourished person will not escape, however, if his nervous equipment has a large element of inherent instability. Some cases are mechanically produced by osteitis, an arthritis, dislocation of vertebræ, or of the sacro-iliac synchondrosis.

PATHOLOGY. A large variety of degenerative and inflammatory changes have been found in the neuralgic nerve, and also an absence of any changes. It is difficult to draw any sharp distinction between neuralgia and neuritis. In some cases the neuralgia is certainly the primary stage of a neuritis, and changes in blood vessels probably play a certain role in the causation of the degenerative condition of the nerves (Gordon). There is often congestion, with or without exudation in the sheath of the nerves. There may be organic disease of the nerve-fibres, or the endoneurium in each case where neuralgic pain is an initial symptom of a neuritis.

Dana sums up the pathology thus: Neuralgia is not an inflammation, but an exhaustion neurosis, with some rheumatic or metabolic irritation of the nerve in addition. There is always, in severe cases, some perineuritis, and exudate into the sheath of the nerve. Starr says that disease of the blood-vessels is sometimes found, and it causes neuralgia by preventing the osmosis necessary for the proper nutrition of the nerve. In many cases where resection of the nerve has afforded us an opportunity of examining it, the nerve-trunk has been found in a healthy condition, proving that the actual causative lesion, in some cases, is certainly not in the nerve, but may be in a ganglion, or in the cortical cells in the brain. This opinion has much clinical backing. A nerve may be resected, and found to be healthy, as when the 5th nerve has been resected for the relief of the pain of tic douloureux, and proves that the lesion is at least as far back as the ganglion. Extirpation of the Gasserian ganglion will relieve some cases of tic douloureux which were not relieved by the resection of the nerve, proving that some cases are actually due to a lesion in the ganglion. Some cases resist even this operation, and prove, therefore, that the cells of the cortex may be the seat of the process. Such cases are rare, and their interpretation is not always clear. In some cases it is probable that the sensory cells of the cortex are diseased, while other cases are classed among the reminiscent neuralgias; that is, the severe pain has made such an impression upon the consciousness, that when the actual disease has ceased to produce the pain, the mental picture is still retained of that area of the body as being the seat of extreme discomfort. Where opportunity for investigation of cranial nerve neuralgias has been afforded, lesions have been very commonly found in the nuclei of the nerves which are located in the substantia Rolandi of the cord and bulb, which is a gelatinous structure, composed of fine nerve-fibres felted together, in which the cells of origin of the nerve-fibres are imbedded.

SYMPTOMATOLOGY. The most obtrusive symptom of an attack of neuralgia is a pain of a burning, darting and shooting character, which is felt running like a stream of fire along the course of a nerve, localizing with peculiar intensity in the area of its distribution. Other nerves in the vicinity are apt to be simultaneously affected, but in a minor degree, and the skin in this area is apt to be red, hot, and shining, particularly if the attack is of long duration. When the pain has subsided, the affected area is left exquisitely tender, but the nerve-trunk, which was acutely sensitive to pressure during the pain, is now insensitive as a rule. Pressure upon the nerve increases the pain during the attack, unless it is so great as to temporarily destroy its function, when the result will be a short respite from the pain. Between attacks pressure upon the nerve is often capable of inaugurating a new paroxysm. The knowledge of this possibility teaches the patient to study such attitudes as will preserve the affected nerve from pressure and extension. These attitudes are somewhat diagnostic. If on examination we find that one gluteal fold is smoothed out, and the patient refuses to allow full extension of the straightened leg, we may assume that he is suffering from sciatica, granting that there has been a history of neuralgic pain, while a persistent lateral flexion of the spine suggests intercostal neuralgia. We often find that the effect upon the spinal segments is so irritative to the motor cells, that it will cause spasm of the muscles in the affected parts. The redness and throbbing of the tissues show that the vaso-motor fibres are directly, or reflexly involved in the process, and according to the area implicated there will be sweating, lachrymation, or salivation. The smoothness of the skin is sometimes mechanical from the swelling of the tissues, and sometimes from trophic changes, which are most distinctively shown in the crops of herpes which are often present, indicating in such cases an implication of the cells of the ganglion. The hair will fall out over a large area, or in patches which distinctly point out the area of innervation of the affected nerve, or it may only lose its color.

COURSE. The developement of a case of neuralgia is usually a matter of only a few hours, but sometimes slight twinges will antedate the actual attack for weeks or months. The duration is an uncertain matter, varying largely with the location. While an attack of neuralgia in the fifth nerve, or one of the brachials, may be expected to subside in a few days, an attack of intercostal neuralgia is a matter of a week or two, while sciatica is more likely to harass the patient for the same number of months, and tic douloureux will generally last his lifetime. The individual paroxysms are usually for a few seconds only, but recur every few seconds for hours, and then subside or completely go away for some hours, when another attack comes on. There is often a clock-like periodicity to the attacks. After the initial attack, there are apt to be others for some length of time, especially if there is any exposure or strain. Night is apt to bring some relief, if the part can be kept warm and the position retained. If this is too uncomfortable to be persisted in, there is usually an attack of pain, and then quiet for a period.

PROGNOSIS. This is always doubtful for actual cure of the condition. Remedies are sometimes to be found which will abbreviate, or terminate the attacks with a fair degree of permanence A small proportion of attacks of neuralgia occur in persons who have no predisposition, but have developed the condition from severity of exposure, or from the combined effects of exposure and a temporary cachectic condition. In such persons the cure of the attack is the cure of the condition, but this is not generally the situation. It may be said that once a neuralgic, always a neuralgic is the rule. This does not mean that all persons will suffer from a permanent tic douloureux who have had an attack of facial neuralgia, or that an attack of sciatica predicates a limping gait through life, but it does mean that that person is very subject upon slight exposure, or strain, or condition of exhaustion to develope a similar attack, so that they may be said to be neuralgic, and have to remember their frailty in all questions relating to the conduct of life. If the patient is young and vigorous, and can give a history of a definite exposure, or some other evident disposing cause, the prognosis is better for immediate relief and future immunity, although we see people of robust developement and healthful lives who are chronic sufferers from some form of neuralgia. The fact is, that while they have a good physical developement, they are nevertheless inheritors of poorly organized nervous cells.

DIAGNOSIS. A typical case presents an intense, paroxysmal, throbbing pain, initially, and perhaps always, confined to a single nerve and its distribution in the tissues. A later implication of the neighboring nerves does not nullify the diagnosis. The nerve-trunk is either insensitive or sensitive only during the attacks of pain, and for a short time afterwards. The pain is in paroxysms, and there are periods in the twenty-four hours when the parts are free from pain, although after severe spasms they may remain sore to touch. The brevity of the pain is characteristic, as is its easy excitability, and in facial neuralgia, for instance, the pain may always be excited by peripheral irritation, such as the touch of a wisp of cotton. There are generally heat, redness, and signs of tension of the affected tissues. It is never bilateral; except when it is the result of pathological changes in or near the spinal column, but may be migratory.

DIFFERENTIAL DIAGNOSIS. Organic nervous diseases like Tabes, Multiple Sclerosis, Tumors of the Brain and Spinal Column, Meningitis, Syphilis, Tumors in the course of neighboring nerves, Aneurysms, etc., may all be marked by neuralgic pain, therefore investigation to exclude such conditions should always precede the diagnosis of any acute pain as neuralgic.

Neuritis and neuralgia are not always capable of differentiation, but typically neuritis is due to an interstitial or parenchymatous inflammation of the nerve-trunks. In neuralgia the nerve is insensitive, when pain is absent, except where it overlies bone, is surrounded by hard fascia, or is superficial. In neuritis the nerve is sensitive to pressure along its whole course, or for a considerable proportion of its extent. The nerve is not swollen in neuralgia, while it is almost always palpably swollen in neuritis. The pain of neuralgia is diagnostic in having periods of perfect remission. The pain of neuritis is constant, although it may have periods when it is much intensified. Neuritis produces muscular weakness, and paralysis soon follows. Later there are atrophy, electrical changes, and anesthesia in the distribution of the affected nerve. Neuralgia is not followed by paralysis or atrophy, except in the late stages of a very severe attack, where it was the sensory precursor of an acute neuritis, and in that case even it is moderate in extent. Instead of anesthesia there are subjective changes of sensation. Neuralgia is apt to be chronic, while neuritis is apt to be acute. Hysteria produces psychalgia, or pseudo-neuralgias. These are pains which are profoundly affected by suggestion and distraction; they do not follow the course of definite nerves in their distribution, and the painful areas are apt to be rectangular, instead of being irregular in outline.

Meralgia paresthetica is the term applied to a disease of the external cutaneous branch of the anterior crural nerve, giving rise to a paresthesia of the anterior external surface of the thigh. It appears to be due to imperfection in the circulation in the nerve, which would pathologically class it among the neuralgias, but it is a paresthesia, and not a pain, which is more like a neuritis. One author states that it is painful only while the patient is in the upright position, but this is certainly not true of all cases.

Melalgia or Erythromelalgia is a painfulness of the extremities only, caused by vaso-motor stasis, producing the pain in the nerves by mechanical compression, combined perhaps with the retention of fatigue toxins, and it is at once relieved by elevation of the affected part.

Herpes Zoster is essentially a hemorrhagic inflammation of the posterior root ganglion, and is accompanied by a severe form of neuralgia, and by a dermatitis.

Referred Pains are similar to neuralgia, in that they are pains distributed over the surface of the body, along definite nerves. Instead, however, of being caused by disease of the affected nerves, they are the superficial expression of disease of some of the internal organs. Inflammations, and irritations of some of the viscera, send impulses along the visceral nerves to their centers in the cord. By the collateral tracts the ganglia in general in that locality are irritated, and among other results we get pain reflected upon the surface of the body. As an illustration: in conditions where the heart muscle is poorly nourished, the sympathetic nerves, which are irritated, affect the 3rd or cardiac ganglion of the cervical sympathetic, which communicates with the ganglion cells of the first dorsal nerve, which is the nerve that supplies the inside of the arm. Hence cardiac disease is liable to cause pain in the chest, and also down the left arm. Such localization has been quite accurately mapped by Head of England.

Epicondylalgia. Pains about joints or bony prominences are often called neuralgias, but Remak calls them Epicondylalgias, and attributes them to strains of muscles, or possibly to a periostitis (Dana).

TREATMENT. While many neuralgic attacks are trivial, and easily alleviated by a well-chosen prescription, there still remains a very considerable percentage which causes extreme suffering, and is most difficult to relieve. It is a matter of the first importance to discover the cause, and especially if it is a physical and removable one like disease of the bone, or joint, or a dislocation. If the attack is not the logical result of some evident exhaustion or refrigeration, we should search for evidences of malaria, rheumatism, syphilis, tuberculosis, diabetes or cancer, any of which conditions are competent to produce severe and long-lasting attacks. We should never be satisfied with our diagnostic technique in an obdurate case of neuralgia until the X-Ray has been employed. A palpable cause may not exist, or the case may prove toxemic, or cachectic, in which case we must look to hygiene and remedies for the cure of the case. In any case the diet should be simple, and so supervised that obesity will be reduced, or anemia corrected. Elimination must be perfect, if we are to obtain good results from our remedies. Hydrotherapy should be employed, in many cases for the relief of pain, and in all cases for the improvement of metabolism. Massage is rarely out of place, but must not be employed too early. The patient's whole life must be regulated; the sedentary should be directed to proper forms of exercise, while the exhausted should be induced to rest. Alcohol and tobacco should be strictly limited, and if possible altogether eliminated, since they both act directly and prejudicially on the peripheral nerves. We are all familiar with the effects of alcohol, and Erb reports that over one half of his cases of intermittent claudication (meralgia) were heavy smokers, and that many cases of intercostal neuralgia, and pseudo-angina can be traced to the same cause. If all forms of medication and treatment fail to afford relief, we must not forget the very remarkable benefit often derived from change of scene. In a work of this character remedies can only be suggested, and the grounds for their employment briefly indicated.

Quinine and Urea Hydrochloride, 1%, subcutaneously will block off sensation, and was of great assistance in giving rest to a victim of herpes zoster, and should be useful under many neuralgic conditions, possibly only in a temporary way. This was used on the recommendation of Dr. Louis R. Kaufman who reports prompt relief in a severe case of lumbago.

THERAPEUTICS. Remedies for the specific types of neuralgia will be found under their respective headings. In suggesting remedies for neuralgic pains in general, Hughes stated that the neuralgia of young and vigorous people is generally rheumatic or inflammatory, and that Aconite, Belladonna, Colocynth and Spigelia are usually curative. Elderly people are generally victims of a degenerative type of the disease, and under such conditions Arsenicum, Phosphorus and Sulphur are indicated. Other remedies commonly de-

manded are Actæa racemosa, Allium cepa, Atropia sulph., Bryonia, Calcarea carb., Capsicum, Causticum, Cedron, Chamomilla, Chelidonium, China, Colchicum, Colocynth, Gelsemium, Kalmia, Magnesia phosph., Mercurius, Mezereum, Natrum mur., Nux vomica, Plantago, Platina, Prunus Virginica, Pulsatilla, Ranunculus bulbosus, Rhus tox., Sepia, Spigelia, Stannum, Staphisagria, Verbascum.

The immediate relief of pain is sometimes all that is needed when the condition is an acute toxemia, and the nerve tends to return to the normal immediately on the cessation of the temporary irritant, and therefore a few doses will cure the case. On the other hand, in some stages of cases of long standing, and great severity, this demand for relief may be so imperative that it must be regarded as of prime importance, although it only palliates a condition which must be cured, if at all, by other means. Morphine stands first in potency, and last in availability, on account of its habit forming tendencies. The coal-tar products are often efficient, and their potency is sugmented by the addition of Codeine or Strychnia. Strych. sulph. alone, in increasing doses, is sometimes very dramatic in its effects, and Thyroid extract has stopped some very obstinate pains. Nitroglycerine, hypodermically, or by mouth, benefits some cases.

Electricity has its strong advocates, and there are those who deny its efficiency. In my own experience I have seen great benefit in some cases. I find that the faradic current helps some patients, while only the galvanic benefits others. I have been impressed, in all modes of application, by the necessity of a very gradual reduction of the current in closing the treatment. In a very severe case of traumatic sciatica, a prompt cure, in the most accurate meaning of the term, was obtained by the use of the faradic current. It was applied as strong as could be borne, for ten minutes, and slowly diminished. At the first renewal of the pain the current was similarly applied. This treatment was kept up night and day for three or four days, and the cure was complete. The current must be mild; 10-15 m.a., especially in facial cases. Some cases respond to vibratory massage, either over the course, or point of exit of the nerve. In the enumeration of curative measures radiant light and heat have a place, and the heat, which is probably the curative agent, may be derived from a battery of lamps in a cabinet, or from some one of the therapeutic lamps now on the market. Heat, plus counter-irritation, is probably the force which we utilize in the high-frequency tube. I have not been greatly impressed with its value in severe cases, and other cases do not demand its employment. Other methods of counter-irritation are sinapisms or the actual cautery to the affected nerves, or painful points. Dotting the surface until it is a rich brown;

instead of stroking, is said to be the most efficient method. Iodine applications have some efficiency, while bleeding locally gives prompt relief at times. Repeatedly freezing the painful areas by Ethyl chloride may relieve, and has been said to cure some intercostal cases. When all such measures have been employed, with little or no benefit, relief from pain can be obtained only by paralyzing the diseased nerve, or by cutting it off from access to the recipient cells in the ganglion, or in the brain. Resection of nerves seems very short-lived in its effects, because of speedy reunion, and therefore dissection of the nerve from its seat, and avulsion has been practised. Next the roots have been resected, and the ganglion removed. Latterly injection of the nerve-trunk has been very extensively practised, and various substances have been injected which will be more specifically described under neuralgia of separate varieties.

Neuralgia of the extremities offers opportunity for the employment of one device which is not applicable elsewhere, viz., splints, or some other apparatus for immobilization of the parts. These devices are not only helpful, but in some cases they constitute about all that is necessary in the way of treatment.

NEURALGIAS OF SPECIFIC LOCALIZATION.

At the risk of a certain amount of repetition, it seems best at this point to speak more specifically of the neuralgias which selectively affect certain definite nerve areas. It is interesting to note that Dana has said in an article in the Medical Review of Reviews that clinically there are only three neuralgias, viz., Trigeminal Neuralgia, Neuralgia of the Brachial Plexus, and Sciatic Neuralgia, and that all the other neuralgic pains were symptomatic or reflex pains, or manifestations of a neuritis, or some injury or irritation of the nerves.

Trifacial Neuralgia, or Tic Douloureux. This is an intense, burning lancinating pain, often agonizing in its intensity. Attacks may come on at first only once in a few months, and persist for a few days. Later the attacks will come more frequently, and as a rule the severity of the attacks increases, pari passu, with the frequency. During the attacks the paroxysms of pain may occur only once or twice a day, or they may come every few moments, or at certain parts of the day with clocklike regularity, the rest of the day being immune, and sleep being undisturbed at night. With the pain often come motor spasms which may not only distort the face, but also pull the head violently to one side. It affects either side indifferently, the supra-orbital nerve most commonly, but it is rarely bilateral, and as rarely does it affect all three of the branches, except in cases of long standing.

When the first branch is affected the pain is supra-orbital, running over the eyebrow, and may extend clear to the vertex. Sometimes pain in the eyeball is the sole manifestation, and in any case the globe is usually tender. When the second, or infraorbital, division is affected, the pain is located over the cheek, implicating especially the malar bone and the antrum. It spreads forward on to the wing of the nose, and posteriorly as far out as the lobe of the ear. When the third or malar branch is concerned, the pain traverses the lower jaw, the tongue, and the corresponding part of the face, but Starr says that while he has seen the pain limited to the tongue, he has never seen taste affected. The attacks, if severe, are accompanied by vasomotor changes, the cheek flushes to a deep red, and the lips, cheek, and eyebrow may be swollen. There may be salivation, lachrymation, and a mucous discharge from the nose. A patient of mine had insomnia as a precursor of an attack, next excessive salivation, and then a paroxysm of pain. The hyperesthesia is extreme, and Patrick has pointed out that if the face is touched with a tuft of cotton on what he terms the "trigger zones," pain will be excited in not only the branch which has been touched, but also in the others. The slightest draft may excite pain, and so a patient is likely to wrap the face up warmly against any possible change of air. Even wiping the nose or eyes may precipitate an attack, and patients sometimes abstain from washing for considerable periods on account of this possibility. Eating often becomes a problem, as the act of moving the jaws is so certain to excite the pain. I had a patient who solved the problem in this way. He first violently blew his nose, which gave such a tremendous shock to the cells of the ganglion (possibly), that they were benumbed for several minutes, during which time he ate as rapidly as he could swallow his food, and then repeated the process. It was excessively painful, but in this way he continued well-nourished, instead of becoming anemic.

Etiology. The etiology and pathology of this disease have been very much in dispute, since autopsies are rare, and examination of the nerve has at times disclosed no evidences of disease, and some have said that the same statement is true in regard to the ganglion itself. This has led to the statement that the lesion was, or might be, as far back as the recipient cells in the brain, and as additional evidence, cases are related where after extirpation of the Gasserian ganglion the pain still persisted. This latter evidence is not competent, as it is most probable that the return was due to imperfection in the operation, or that the pain was psychic, what is often termed reminiscent pain. On the subject of etiology, Patrick says: "Tic Douloureux is a clinical entity of unknown pathology, and while a large number of cases can be found to have had sinus disease, pyorrhea, or bad teeth, I have never known a case to have been cured by the removal of the teeth, or by the relief of any disease of the sinuses, and it is certain that arteriosclerosis, and degenerative changes, are not competent to produce it, nor is it an auto-intoxication, although we cannot prove it. It is never found in Bright's Disease, or diabetes, and alcoholism and lead poisoning have no apparent influence on the disease. There is nothing to support the theory that it is due to changes in the Gasserian ganglion." On the other hand, Starr says that it is caused by vacuolization and atrophy of the neuron bodies in the Gasserian ganglion, with secondary degeneration of the other axons, and a connective tissue inflammation in the ganglion, and also an endarteritis in the small vessels in the ganglion. The same changes have been found in intercostal neuralgia and tabes. Excision of the ganglion has never failed to cure if properly done. Dana says that it is a degeneration neurosis, and rarely occurs before the forty-fifth year; that it is a kind of trigeminal angina; that the neuralgia is due to an obliterating endarteritis, causing deficient nutrition in the nerve, and is accompanied by vascular spasm; there is an irritative and atrophic process occurring in the nerve and its ganglion. He points out, on the other hand, that there is a symptomatic tic douloureux, which is a disease of early life; that it is a trifacial neuralgia, and he does not believe it should be the subject of operation. Deaver says that the general predisposing causes of tic douloureux are a neuropathic diathesis, exhausting maladies, loss of blood or tissue-juices, anemia, cachexias, senescence, and arteriosclerosis. Cold, exposure, overexertion, and mental strain act similarly. The mineral poisons, toxins of diabetes, gout, Bright's Disease, etc., are all included. The various infectious diseases, abscesses along the course of the nerve, and carotid aneurysm may cause it. Within the skull it may be caused by fractures along the base, tumors of the ganglion, or cholesteotomata near the ganglion, tuberculous and syphilitic granulomata, and these conditions should be kept in mind as possible causes of the trigeminal pain.

DIAGNOSIS. The tender points are at the supra-orbital foramen for the supra-orbital branch, the nasal foramen at the upper part of the nose for the supra-maxillary branch, the infra-orbital foramen below the upper eyelid, the malar for the inferior maxillary branch, the temporal in front of the ear, the labial on the upper lip, the mental, which is the place of exit of the mental branch on the jaw. Another peculiarity is that an irritation in the distribution of any one of the three branches of the trifacial nerve may start a pain in another branch. These areas are called "trigger zones."

The tender points just enumerated demonstrate that there is a neuritis, or a neuralgia of the trigeminal nerve. If it is a simple neuralgia and acute in type, prompt recovery will be the rule. If it becomes chronic, its moderate severity, and its evident etiology will serve to distinguish it from tic douloureux, for that has no clinical distinction aside from its persistency, and the developement of a definite symptom-complex peculiar to the given case, yet conforming to a general type.

PROGNOSIS. This disease has been said to be incurable; to be positively remedied only by the removal of the offending structures. It is intractable and obstinate, and in some cases possibly incurable, but I think that homeopathic medication is competent, perhaps with the aid of electricity and other adjuvants, to cure some of the cases, for it must be understood that there is danger to life in the radical operations, and that if there is no fatality, yet very severe physical disabilities may result.

TREATMENT. In all cases the hygienic care, outlined under general treatment, should be instituted, and scrupulously carried out, if we are to reap the results which are reasonably to be expected from the action of a properly selected remedy. In such an intractable complaint the number of suggested remedies is almost endless, nad this is true of both schools of medicine, This usually means that no remedy is efficient, but I think that it is not the truth in this instance. They should be tried before operation is invoked, and certainly should be pains-takingly employed if operation is impossible, or is refused. Aconite has appreciably helped several of my cases, and with electricity as an adjuvant has produced in two cases such long remissions, and such tractable relapses that a permanent cure is certainly suggested. Hughes reported favorable results from Arsenicum, and said that Phosphorus and Sulphur are worthy of consideration. Other remedies employed with some measure of success by competent practitioners in facial neuralgias of all types are Actæa racemosa, Belladonna, Bryonia, Chelidonium, Colchicum, Colocynth, Gelsemium, Gnaphalium, Kalmia, Kali bichromicum, Lachesis, Magnesia phos., Mercurius, Mezereum, Natrum muriaticum, Natrum sulph., Nux vomica, Platina, Pulsatilla, Rhododendron, Spigelia, Verbascum, Viburnum.

Analgesics and counter-irritants may be needed to tide the patient along until specific medication has had time to produce its effects. In many cases these measures fail to give relief, and surgery of some sort must be invoked. Formerly, resection of nerves was extensively practised, but now has been practically abandoned, on account of the speedy reunion of the separated parts. Another method is the avulsion of the nerve, as suggested by Van Gehuchten and others. Van Gehuchten states that if a nerve is gradually pulled until it tears out, the resulting shock destroys the cells in the ganglion. Laplace, of Philadelphia, reported four cases treated by this method, with apparent permanent relief (N. Y. Medical Journal, Dec. 9th, 1905). The most popular device at present is the injection of the nerve with some paralyzing agent which cuts off sensory impressions, and yet does not cause motor paralysis, nor sloughing of the tissues. This treatment was first suggested by Schloesser, of Munich, in 1903, but was first reported by his student Oswalt in 1906. It was popularized in France by Lévy and Baudouin, and in this country by Patrick, Killiani, Hecht, Gordon, and others. The technique of administration may be found described by Patrick in the Journal of the American Medical Association for Jan. 20th, 1912. It consists of the instillation into the nerve-trunk at the foramen of a 75-90% solution of alcohol, which substance, according to Schloesser, when brought into contact with the nerve, produces a degeneration of all parts except the neurilemma. Experience has shown that if it is deposited near the nerve there may be an immunity from pain, but that it will be only for a month or two, but that if it has been deposited in the nerve, or nerve-sheath, there will be relief from pain for a long time, up to four years, perhaps, and it may never return. Bodine and Keller use 2 c.c. of the following solution in each branch: Cocaine hydrochlorate 1 grain, Chloroform 10 minims, Alcohol 3 drams, Distilled Water q. s. to make a half ounce. Killiani uses 1 to 4 c.c. of an 80% solution of alcohol, and believes that Schloesser's method guides one with absolute accuracy to the nerve, but that the method of Lévy and Baudouin is easier. As has been said, the relief, while prompt, is usually strictly limited in duration, and the first success may not be repeated. I have a patient who has been repeatedly injected by very competent men in various countries, with only the most temporary relief. In such cases some operation on the ganglion, or near, it is demanded. Removal works a certain cure if properly performed, but there are few operators who have been able to reduce the mortality to tolerable limits. It is, on this account, recommended only for the young and robust, while the aged and delicate should look to injection of the nerve for their possible measure of relief. To avoid the dangers of the tremendous hemorrhage, and the probable injury to the frontal lobes of the brain from the necessary handling, Abbe devised another operation. He divided the first and second roots just distal to the ganglion, and capped the ends with rubber tissue to avoid the possibility of reunion. The sensory root has been divided just behind the ganglion.

Cervico-Occipital Neuralgia. This kind of neuralgia, in slight degree, is experienced by almost every person at some time in his life. Pain in the back of the neck and head is very common, when the muscles of the neck are continuously strained, as by dentists, or when there is congestion of the membranes of the brain from emotion, overwork, overstrain of the eyes. It is a common result of new growths, or structural changes in the posterior fossa. As a specific complaint it is due to intoxications and infections affecting the four upper cervical nerves, but the great occipital most generally. The pain is usually bilateral, but may be on one side only, and a dull tiresome ache is the rule, but it may be very sharp and excruciating. Diagnostically there is tenderness between the mastoid process and the first cervical vertebra, and over the second and third cervical spine, and on the vertex. The attack usually lasts five or six weeks.

DIAGNOSIS. It is diagnosed from migraine from the fact that that is unilateral and does not limit itself at the vertex, but goes over onto the forehead; it is periodical, and it not paroxysmal. Neurasthenics have such a pain very commonly, but it is tired and aching in character instead of being deep and boring. The pains of hysteria are shifting, as are those of spinal irritation.

TREATMENT. The principal remedies are Belladonna, Bryonia, China, Mercurius, Nux vomica, Pulsatilla, Ranunculus, Rhus tox., Sanguinaria, Sulphur, Veratrum alb. Locally hot and cold applications, freezing by Ethyl chloride, counter-irritants as mustard, iodine and the actual cautery are useful. Blood-letting has its very strong advocates. The injection of alcohol, sterile water, normal salt solution, or cocaine into the nerves is the most reasonable adjuvant treatment at the present time. The great occipital nerve has been resected with slight benefit.

Brachial Neuralgia. Oppenheim has called attention to, and described a brachialgia, which he says is relatively frequent. It is a unilateral diffused pain in the arm, and does not follow definite nerve tracts. It developes upon a neuropathic basis, and is usually accompanied by other symptoms. There is a frankly hysterical variety, which is accompanied by spasmodic jerkings of the arms. It must be diagnosed from neuralgia, neuritis, and spinal affections, as well as from diseases of the bones, vertebræ, joints and muscles. It is suggested that the so-called reflex neuralgias, often accompanying facial injuries, may be of the same nature, and must be remedied by treatment of the basic neurasthenia. On the other hand, the various nerves of the brachial plexus are occasionally the seat of a true neuralgia, which is about as frequent as facial neuralgia, or sciatica. This has been briefly described by some authors, and ignored as an entity by others, but has been most exhaustively treated by Dana (Medical Record, Feb. 23rd, 1907).

Brachial Neuralgia. It is liable to affect the plexus generally, and the nerve roots, but it may be limited to the terminal portions of individual nerves. The less severe cases will exhibit a pain which is limited to the distribution of the circumflex nerve to the bend of the elbow, or to the nerves about the wrist, or of the palm of the hand.

These cases very commonly result from irritation by growths on the spine, or joints, or pressure of new tissue. Heredity plays its part, but it is most usually found in females of middle age, who have led easy lives, and who have been subject to some injury, or occupational strain. It is often attributed to rheumatism, but rheumatic remedies are not efficient in its treatment.

It is the result of an irritant acting upon a neurosis, and in severe cases there probably is always some exudate in the sheath of the nerve. If glossy skin developes, if there is atrophy of muscles, and there are trophic changes in the finger-nails, the malady in question is no longer a neuralgia, but a neuritis. It is distinctively a disease of the upper cords of the brachial plexus arising from the fourth to the seventh cervical segments, with the exception of the internal cutaneous, which arises from the lower cord. The collateral branches are mainly muscular, and so we must look for the source of pain to the terminal branches, which are sensory-motor. These are the circumflex, radial, musculo-cutaneous, internal cutaneous, median, and ulnar, and they convey cutaneous and muscular sensation to the shoulder, as well as to the arm.

SYMPTOMATOLOGY. The pain usually begins suddenly, often on awakening. It is generally deep and diffused, and not darting, usually in the upper arm and shoulder, less often or subsequently in the forearm and fingers. It is usually worse at night, probably from posture, and from stretching movements of the arm causing tension on the plexus, the effect of which is shown by the reduction of the elbow-jerk in brachial neuralgia, just as the Achilles-jerk is reduced in sciatica. There is no objective change in the limb at first, but later a vasomotor loss may be disclosed by a slight swelling of the arm and fingers, which are tender and sore. Shoulder-pains in general point to disease of the circumflex; when they are superficial they are felt more posteriorly, and over the body of the deltoid, with a painful spot where the nerve becomes superficial. The deep pains are not only in the shoulder, but extend also down into the fingers, usually the first, second and third, or the second and third only. This distribution is due to the fact that from the posterior cord of the plexus come both the circumflex, and also the radial. Pains just above the shoulder, and about the acromion, are due to involvement of the supra-acromial nerves which arise from the fourth cervical segment. Pains on the anterior and inner surface of the shoulder. and the upper arm are from the intercosto-humeral. Just above the origin of the circumflex arise the collateral branches to the subscapular and latissimus dorsi, and thence the pains radiate back into the region of the scapula. Upper arm-pains are often associated with interscapular-pains at a point a little lower down than those found in the shoulder-pains, viz., in the region supplied by the first and second dorsal. The tender points are along the inside of the arm, midway between the inner condyle of the axilla.

The sensory fibres from the same segments as the circumflex and radial, viz., the 5th, 6th and 7th cervical, when involved, cause localized pain just above the interscapular space, at the level of the first and second dorsal vertebræ. The pains of brachial neuralgia are most prominent on the inner side of the biceps, and extending to the axilla, and are the result of disease of the internal cutaneous, and the musculo-cutaneous, when they are superficial. Anteriorly the parts are supplied by the musculo-cutaneous, which also sends a branch to the periosteum, and explains the deep-seated pains. They may be due to some extent also to inflammation of the muscles, and cellular tissue. The outer side of the arm is supplied by the radial both superficially and deeply, and in general it is true that if pains extend downward they are due to affection of the radial nerve. Genuine neuralgia in the forearm is rare. We have ulnar pains which follow the course of the nerve, and radial pains which are characteristically on the back of the arm to the fingers. Median pains are the least frequent. Sometimes the whole hand is involved in neuralgic pains, but in this case they are generally hysterical. When the hands and fingers exhibit paresthesias and local pains, they are usually due to local disease, and not to neuralgia.

DIAGNOSIS of Brachialgia cannot be made alone from the localization of pains in the arm. It is rarely double, and when so is an exhaustion neuralgia, and not a neuritis. Pains in this locality are far more often the expression of disease in the bones, or from vascular derangements. Injuries to the plexus may be a cause, but they are far more commonly due to diseases of the spinal column, menigo-radicular diseases in the spinal canal, and new-growths, or degenerations, in the cord itself. Schwab points out that a pain in the elbow is often an indication of Pott's disease of the cervical spine. An X-Ray examination should always be made in severe or protracted cases. In all these neuralgias we find tender spots, called the points of Valleix, but Dana says that they are not always classical in location, and agree in a general way only with the distribution of the cutaneous nerves. In Occipital neuralgia they are well-marked over the second and third cervical vertebræ. In Cervico-brachial neuralgias they will be found down almost to the first and second dorsal spines, while Brachial neuralgias will involve those spines. In the arm tender spots will be found where the musculo-cutaneous, the lesser internal-cutaneous and the cutaneous branches of the radial emerge from the muscles.

PROGNOSIS is for cure under any treatment in from three to six months, and a recurrence in about ten per cent. of the cases.

TREATMENT initially is rest, either in a sling, or by the use of a splint. Next we should make use of heat by the continuous electric pad, or dry or moist packs, or by the electric bath, or the leucodescent lamp. Massage or electric currents are not advisable until the acute stage has passed, perhaps after four or five weeks, if the case lasts that long. Some of the lasting pains are reminiscent or habit-pains, and may be ignored. Physiological medicine does not act as might be predicted, as Dana says that the salicylates are not very useful, and if given must be used in large doses.

Homeopathic remedies usually relieve the actual neuralgic cases promptly. Those suggested are Rhus, Veratrum alb., Bryonia, Mercurius, Nux vomica, Pulsatilla, and Sulphur. I have had success with these, and also with Sanguinaria and Ranunculus.

Intercostal neuralgia is a very common form of pain affecting the intercostal nerves, but one which must be differentiated with a great deal of care from pains in this locality due to disease of the vertebræ, diseases affecting the roots of the spinal nerves, and the so-called referred pains which are the superficial expression of visceral disease. It is much more common in women than in men, and may result from a large variety of infections and intoxications, and often follows a bruise or a strain. Smokers of tobacco are liable to a great variety of these pains, and they may be also the result of the sudden cessation of the habit. I have frequently seen it follow a severe attack of coughing, when experienced by a person whose resistance has been lowered by infection or anemia. Most cases are from "taking cold," and so the complaint is more common in the colder months of the year. The pains are sharp and stabbing, or run about the chest like streams of fire, and are practically always aggravated by motion, or by the strain on muscles produced by lying long in one position.

The diagnosis often has to be made upon lines of exclusion, but on the positive side we depend upon the discovery of the peculiar painful points, of which there are three, but not all of them are to be found in every case. The first is at the sterno-costal articulation, the second in the axillary line, and the third near the spinous processes. The X-Ray will disclose disease of the spine; lumbar puncture will in many cases determine the presence of diseases giving rise to root-pains.

It is diagnosed from Pleurodynia from the fact that the latter is a more wide-spread pain, and has no definite localization of painful spots. Mastodynia is directly confined to the breasts, which may be indurated, and may even produce some colostrum. Among the referred pains the most essential for differential diagnosis are the cardiac. These pains are rarely distributed to the arm except in neurotic persons, but there is no sign by which we can certainly differentiate them from intercostal neuralgias, except by the discovery of the presence or absence of physical reason for the pain.

Some cases are very obstinate, and are relieved only with the cure of the underlying neurasthenia, removal of some bony deformity, or avulsion or resection of the nerve if there is degeneration of the cord. Strapping of the chest is beneficial in some cases, while in others we find freezing repeatedly at the exit of the nerve-roots (with Ethyl chloride) all that is demanded. The actual cautery, brought to a white heat, and lightly dotted along the side of the spinous processes, is probably the most commonly efficient measure. Iodine, Cantharides and mustard are of service at times. Electricity in all its modalities has given relief; stabile galvanism, with a very mild current, has been my choice. Remedies usually are competent to produce a cure.

I have found Arnica to be indicated in the majority of cases, as a neuralgic tendency is so often localized by a strain or shock. Ranunculus and Spigelia have also been clinically verified. Other probable remedies are Aconite, Actæa racemosa, Anantherum, Bryonia, Causticum, Chelidonium, Colchicum, Gautheria, Kali carb., Nux vomica, Pulsatilla, Rhus radicans, Rumex crisp., Senega.

Herpes Zoster, Zona, or Shingles. This is an acute infectious disorder, due to inflammation in a root ganglion, producing neuralgic symptoms along the course of a nerve, possibly anywhere on the surface of the body, and terminating in a localized dermatitis. It is generally sporadic, but sometimes is epidemic, and it is most likely to occur in spring and fall. It usually affects persons between the 15th and 30th year, but aged persons are rather prone to attacks. It would seem to be from some specific infection, since one attack seems to confer a degree of immunity, and a second attack is very unusual. It has been attributed to rheumatism, gout and syphilis, and poisoning by arsenic and carbonic acid gas, and intestinal intoxications. Malaria, typhoid, meningitis, pneumonia, tabes dorsalis, and paretic dementia are also accompanied by, or are the cause of, attacks of herpes. The Gasserian ganglion is especially vulnerable, and also those on the 3rd dorsal to the 2nd lumbar, which are in relation to the gastro-intestinal tract by sympathetic afferent fibres. Whatever may be the infection the posterior root ganglia show a peculiar susceptibility, and usually only one ganglion is affected, but the

2nd, 3rd and 4th cervical are commonly attacked, and possibly two or more roots in other parts of the body. From this selective invasion it has been termed posterior poliomyelitis.

The pathology is based upon the researches of Head and Campbell (Brain, Dec., 1900). There is an acute inflammation of the ganglion upon the posterior root. There is an extravasation of blood, and an exudation of small round cells, a destruction of the fibres and cells, and an inflammation of the sheath of the ganglion. Next an acute degeneration of greater or less extent appears, followed at times by a secondary sclerosis. In a few cases an acute hemorrhagic inflammation extends from the ganglion into the peripheral nerves, where the acute degenerative products are similarly observed, and a secondary degenerative sclerosis ensues. By the tenth day the posterior columns of the spinal cord may exhibit a degeneration. The vesicles in the skin contain a serum, which is perfectly sterile, and the inflamed lymphatics in the neighborhood contain no bacteria.

SYMPTOMATOLOGY. The attack may be ushered in by a localized hyperesthesia, which may precede the eruption for months, and persist with equal stubbornness after it has passed away, but usually there is a fever and malaise for a few days, followed by pain, soreness and burning in the area of distribution of a given spinal segment. This pain may be agonizing, but in any case, after a few days the affected area is covered by discrete groups of papules on a reddened and swollen base. In a few days these become vesicles with turbid and purulent contents, and in one or two weeks they dry up, forming a brownish crust, and by the end of the second week they fall off, leaving reddened or pigmented patches, and occasionally scars. If the eruption is on the head and face, it follows the distribution of one only of the three main branches of the 5th nerve. The location is usually at the point of the highest efficiency of the nerve area, and causation will reveal the definite nerve leading to this area. On the trunk the eruption does not follow the distribution of a nerve, but the innervation of a spinal segment, and on the extremities is distributed after the same manner, but longitudinally. It does not follow peripheral nerves, and their cutaneous distribution, unless such nerves are injured or inflamed. In folklore it was said that if bilateral it was fatal, which had in it an element of truth, since a bilateral distribution indicates a serious disease of the vertebræ or meninges.

DIAGNOSIS is from pleurisy, since the pain may be so sharp as to resemble it, but auscultation will reveal the facts. It resembles superficially vesicular eczema, and simple herpes, but the antecedent pain calls attention to a difference at the outset, the distribution of the eruption is diagnostic, and the comparatively prompt termination of the disease stamps it as an acute infection.

PROGNOSIS. Generally the patient recovers fully in two or three weeks. As has been said, it may be anticipated by a hyperesthesia, which persists long after the eruption has disappeared. In the same way, the neuralgia may remain as an obstinate condition, and this is especially common in senile cases.

TREATMENT. In the first place there should be surgical cleanliness. Dry antiseptic dusting powders like Oxide of Zinc, and starch powder are helpful, and especially so, if over them is placed a wide muslin binder to keep the area from the friction of the clothing. It has been advised to paint the eruption with Collodion to subserve the same purpose. As a local anodyne, Boric acid with Cocaine 1% will often give relief for the burning sensations. In any case a free action of the bowels and kidneys should be preserved, since many cases are bound up with some intestinal putrefactive process.

THERAPEUTICS. Pierce advised Aconite for the perivesicular stage.

Dearborn advises Belladonna for the same stage, with an unwonted redness of the skin, extreme sensitiveness to contact, worse from warm applications. He also advises Spigelia at this stage for radiating pains relieved by rest, warmth, and pressure.

For the vesicular stage, Graphites when left sided, with large vesicles and excessive burning, aggravated by warmth, and relieved by washing the parts. Hypericum for vesicles beginning with sore places, and forming hard yellow crusts, associated with severe sticking, smarting, and occasional sharp neuralgic pains. Useful internally and locally in the 2x. Cistus Can. when worse on the right side, with extreme sensitiveness to cold. Kali brom. in protracted cases, after the neuralgic pains have ceased. Kalmia, Mezereum, Ranunculus bulb., for facial neuralgia after the eruption has passed away.

Rhus, Arsenicum, Cantharides, Dolichos, Mezereum, Prunus spin. are also among the long list of efficient remedies.

Lumbar Neuralgia. The branches of the lumbar plexus are not commonly the seat of a true neuralgia, but are very liable to be compressed by new-growths in the abdomen and pelvis, and by diseased enlargements of the kidney. When they are the seat of a genuine neuralgia, it is from the common constitutional causes of neuralgia; and especially at this point it may arise from diabetes.

Lumbo-abdominal Neuralgia affects the two upper lumbar nerves, which are almost entirely sensory. There is a bilateral pain in the lumbar region, while on one side only, as a rule, there is pain in the abdomen down to the scrotum and spermatic cord, which may involve the testicles and penis. During the attacks, the abdominal muscles become contracted, and the skin may be paresthetic, but in old cases it becomes anesthetic generally. Trophic and vasomotor disturbance (herpes) may be present, and spontaneous ejaculations may occur.

Obturator Neuralgia, a pain along the internal aspect of the thigh, is often associated with obturator hernia.

Crural Neuralgia gives rise to a pain which extends along the course of the crural nerve to the inner side of the leg and the foot. The tender spots are found in the inguinal region, on the inner surface of the knees, of the internal malleolus, and the inner border of the foot.

For all these types the indicated remedies are Arsenicum, Conium, Gnaphalium, Hamamelis, Iris versicolor, Rhododendron, Salicylic acid, Spongia, and Xanthoxylum.

Sciatica. This is a form of neuralgia affecting the sciatic nerve, and giving rise to an atrocious pain running as high as the waist in some cases, and extending to the ankle. The pain is always severe, and sometimes unbearable, and is generally protracted, yet with a tendency to recovery.

ETIOLOGY. It comes from a variety of infections and intoxications. Gout and rheumatism are the most common causes, excited by exposure to cold and wet, especially if aggravated by heavy lifting-strains, or by occupations involving protracted standing. Alcoholism predisposes one to attacks, it is very frequent in diabetics, and may follow any one of the infectious diseases. In my experience, it is seen with great severity after typhoid. Without an evident cause, there should be suspicion of a disease of the pelvis, or of its contents, or of the lower spine, and Goldthwaite, of Boston, considers subluxation of the sacral joint to be a very common cause of the complaint. Some such cause is always to be suspected in the cases of double sciatica, for except in diabetic cases, the double variety generally is found to be due to disease of the spine or meninges.

PATHOLOGY. The pathology has been considered to be the same as that of other neuralgias, except that there is always some degree of neuritis associated with it in the severe cases, or those of long standing. Post mortem records are rare, and therefore Hunt's report of an autopsy, together with the study of fifteen other cases from recent literature, is most useful. He found the sciatic nerve larger than normal, from the presence of a firm, homogeneous, translucent deposit in the perineurium. Some of this material was also deposited in the popliteal space. Microscopical examination showed a thickening of the connective tissue along the course of the arteries of the sheath, which were markedly sclerosed and thickened. The nerve-fibres were free from deposits, and there was no infiltration of round cells, nor any signs of acute inflammation. From literature he found that in many cases there was a fine powdery deposit of urate of soda, such as is found in the joints in gout, and that this gelatinous deposit seemed to indicate the same condition. He divides sciatica into two groups; first, the neurotic, with slowly developing symptoms; and secondly the neuritic, with inflammation of the sheaths, and of the nerve-fibres, and says that most cases belong to this latter class.

In old cases which have come to operation, all grades of inflammation are found in the nerve-trunk, and most extensive adhesions are found between the nerve and its sheath.

SYMPTOMATOLOGY. The attack is often ushered in by a sore lameness, with some weakness, as if it were a neuritis, but in the majority of cases there is a sharp shooting pain, which is often described as similar to a stream of boiling water running the whole length of the leg. When fully established, it extends down the back of the thigh to the knee, and thence down the outer side of the leg to the external condyle of the ankle, and into the dorsum of the foot. It may extend up as far as the waist-line, if the upper branches of the nerve are involved. The points of maximum intensity are at the sciatic notch, and down the back of the thigh to the knee at the popliteal space. The tender points are above the hip-joint near the posterior iliac spine, at the sciatic notch, at the middle of the thigh, behind the knee, below the head of the fibula, behind the external condyle, and on the dorsum of the foot. Gara found that in 118 out of 124 cases, a tender spot could be found by pressing the finger into the abdomen at a point one finger's breadth below the umbilicus, and two fingers toward the affected side. The pain from such compression radiated down the sciatic nerve clear to the toes. The pain is deep, and never superficial, with paroxysmal exacerbations, when it is unbearable. In some cases the pain comes in shocks. It is curiously varied in its aggravations. As a general truth it may be predicted that any force which will extend the nerve will aggravate the pain, but it is not always true. Sometimes a rigid quietude of the limb will produce almost perfect comfort, yet at times the only relief is from relatively severe exercise. There are few sensory changes on the surface, but paresthesias may be quite pronounced. In cases of severity, and in milder cases of long duration, the limb will show atrophy, glossy skin, and very rigid contractures, from which the individual may be disabled for a long time. Herpes is a common complication.

PROGNOSIS for ultimate recovery is good in even the most severe and contracted cases, although the patient may suffer from some degree of pain for a long time. In cases of moderate severity, recovery may be expected in a month or six weeks, but some cases last from a few months to one or two years. Relapse is very frequent, but it is usually lighter than the original attack.

DIAGNOSIS of sciatica depends first upon exclusion. A pelvic examination will exclude tumors, and displacement of organs in that locality; physical examination, supplemented by the employment of the X-Ray, will assure us of the condition of the bony skeleton; while lumbar puncture will detect the presence of a meningitis, and tabes dorsalis. Affections of the cauda equina give rise to similar pains, generally bilateral, but above all, diagnostically, give rise to urinary incontinence, bowel disturbance, and anesthesia over the coccygeal area, which are not present in sciatica. Some cases of bursitis are confusing. On the positive side of diagnosis we have Lasegue's sign, which is this: when the whole leg is hyper-extended and flexed toward the body, it causes a characteristic pain, shooting from the foramen of exit in the sacrum down toward the heel, with a marked co-incident spasm of the muscles. When the patient is stripped, and made to stand upright, the gluteal fold is found to be smoothed out on the affected side, the thigh is somewhat abducted, and the knee is slightly flexed even when standing in an upright position. This diagnoses the case from one of rheumatism. Bonnet's sign is an increase of pain when the affected thigh is abducted against the resistance of the unaffected thigh. Martin's sign is pain on flexion of the affected thigh against the resistance of the unaffected one. Patrick's test for differentiating sciatica from arthritis is as follows: The patient lies flat on the back, with one leg extended, and the ankle of the other side placed just above the knee of the extended leg. With the ankle in this position, the knee is then depressed toward the bed or couch: that is the thigh is abducted externally, rotated, and extended at the same time. In arthritis this will nearly always cause pain, and there will be found to be a restriction of this movement.

TREATMENT. In so painful and obdurate a disease it is not surprising that there should be a wide range of remedies, and some very extraordinary methods of treatment. Some of these are worthy of presentation, while others are examples of ingenious industry, designed to satisfy anxious friends, and impatient patients, but of doubtful benefit in the final analysis. While we do not cure all of our patients quickly, nor commend our methods to all of them, I feel that we rarely need more than a modicum of analgesics to tide us over the hard places, while a fairly prompt and thorough cure is being worked by our remedies. However, treatment of this malady consists in more than the administration of medicines, and the following adjuvants may be of service. Ironing the nerve, either with the old-fashioned flat-iron, or the Oudin high-frequency electrode is distinctly of service, as is also hot water, directed with pressure along the nerve. Danneh1 treated severe cases by seating the patient in a hot bath, with the foot of the affected leg braced against the foot of the tub, the water being at 100-104° F. The patient grasps the side of the tub with both hands, and pulls the trunk forward and over, thus stretching the nerve. He remains in the tub 30-40 minutes. A hot flax-seed poultice is mussy, but often comforting, and has a psychic effect from tradition. A wet pack is always good, and Leszinsky has suggested this method. Take the leg of a pair of trousers (a doubled drawer-leg would be better), dip it in water at 65° F., and draw it over the leg; cover it with a roller bandage, which transforms it into a continuous pack during the whole night. Take this off in the morning, and give the limb a warm bath, followed by massage. These measures will be about all that is necessary in the average case, as adjuvant to proper medication, but some cases are severe from the beginning, and assume chronicity. Weir Mitchell gave prominence to the method of treatment by the long splint. Both the hip and knee joint must be immobilized. The limb is smoothly bandaged from the hip to the knee with a flannel bandage; a long splint is applied, giving a slight bend to the knee, and the heel is carefully supported. The splint is left on for three days, when it is taken off, the limb massaged, and the splint replaced. This is repeated daily until the patient has been free from pain for some days, when it is only applied at night for about a week. Then it should be removed, and the patient should for a time either stand or lie; he should not sit down, and thus bring a severe strain upon the nerve. If the case is already chronic when first seen, it is a matter of judgment whether or not the patient should be anesthetized, and the adhesion of the nerve to the sheath broken up by over-extension of the limb, before the application of the splint. I have found that a plaster cast presents some points of advantage over the splint. When over-extension has proved futile, other means have been advocated. One of the older methods is to make an incision just above the popliteal space, between the semi-membranosus, and the semi-tendinosus, hook up the nerve, and making traction toward the trunk, and not toward the extremity, to avoid tearing off the peripheral branches, lift up the leg by the nerve two or three times, put back the slackened nerve, and sew up the incision. For the same purpose, Pers, of Copenhagen, dissects out the nerve up to the sciatic notch, opens the sheath, and frees the nerve from its adhesions. When the nerve is much swollen, and very tender, a long ordinary needle, under strict asepsis, is inserted into the swollen sheath, and left in situ for an hour. This drains out the serum. Various injections have been made about and into the nerve. 30 c.c. of normal salt solution, at freezing temperature, may be injected alongside the nerve daily. Plain saline, or sulphate of magnesia 4%, or plain distilled water, with or without cocaine or morphine, have been injected subcutaneously over the nerve in quantities of 50-100 c.c., for the mechanical pressure and analgesic effect. A solution advised is Cocaine 1/100 gm., Chloride of Sodium 21/100 gm., Morphia 2/100 gm., Distilled water 100 gm. Deep injections of Chloride of Sodium, or Sulphate of Magnesium 4% have given better results when injected with a long needle at a depth of 4-5 cm., in quantities of 40-60 c.c. daily for five or six days. The point of injection is at the junction of the inner third with the outer third of a line drawn between the sacro-coccygeal articulation and the postero-external border of the great trochanter. Leszinsky injects from 80-120 c.c., normal salt solution at the ischiatic notch, repeating it from three to six times, three being the average. He claims that penetration of the sheath is unnecessary and dangerous. Alcohol has been injected in and near the nerve, but atrophy and trophic disturbance have followed its use. Nitrate of Strychnia gr. 1/60th has been injected over the nerve, and into the buttock, with prompt benefit. In desperate cases epidural injections have been advised. They are made in the sacro-coccygeal space, which in adults begins at the lower edge of the first sacral vertebra, and extends to the sacro-coccygeal articulation, and at this lower level the injections should be made. Strauss advises injections here of from 50-60 c.c. normal salt solution, and says that it is diagnostic, since failure to relieve pains by this means shows that they are not sciatic in origin. Electricity has been serviceable. Stabile galvanism has been my choice usually, making use of a strong current, but faradism has at times been efficient. The greatest benefit has seemed to come from the employment of a current strong enough to substitute electric-pain for nerve-pain, and reducing the current, after 15-20 minutes, very slowly to normal. Oudin high-frequency seems to have advocates, and many get good effects from radiant heat, the leucodescent lamps, and the electric cabinet. The hot air baking methods are of service, and also the mud baths.

THERAPEUTICS. From the large number of possible remedies some one of the following would be most probably indicated, viz., Aconite, Arsenicum, Colocynth, Ammonium mur., Belladonna, Bryonia, Gnaphalium, Ignatia, Lycopodium, Rhus tox., Sulphur, Calcarea carb., Chamomilla, Arnica, Gelsemium, Iris versicolor, Kali bich., Kali carb., Kali iodide, Natrum mur., Nux vomica, Phosphorus, Plumbum, Pulsatilla, Salicylic acid, Sepia, Silicea, Tellurium, Valeriana, Veratrum, Xanthoxylum. Hughes would favor Chamomilla, Ignatia or Colocynth in a young person, and an interstitial type of case, but would use Arsenicum, Lycopodium or Plumbum in an older patient, and a more parenchymatous involvement. O'Connor advised Gnaphalium where other obvious indications were wanting. Bartlett inclines toward the employment of Aconite and Gelsemium in an acute case.

Coccygodynia is a neuralgia which is characterized by a pain in the coccyx which may be spontaneous, or may not be, but it is sure to be excited by any movement which brings a strain upon the muscles and ligaments of the part. It is almost exclusively a disease of the female sex, it is a common symptom of neurasthenia, it is often excited by local injury, and (whatever is the truth) the larger proportion of cases are ascribed to this cause. The underlying cause is generally neurasthenia. Pathologically it is the neuralgic response to an inflammation of the ligaments of the coccyx, either directly from injury, or indirectly in the case of the neurasthenic. In the latter instance the muscles are relaxed, and the ligaments, on account of lack of muscular assistance in preserving posture, are exposed to a continuous and unusual strain, which induces inflammation, and pain results.

SYMPTOMATOLOGY. As has been said above, the pain may be spontaneous, and while rarely acute, it is nagging and exhausting. Whether spontaneous or not, it is always aggravated by muscular strain, and hence it pains the patient to sit a long time, or in some cases to walk, although that may be a method of relief. Defecation and micturition may bring distress. The pain may be a sudden piercing one in the coccyx, or a rending sensation as though the bone were being bent backward. It may only be the unpleasant sensation of a chair or couch being too hard, or there may be a drawing sensation along the coccyx down to the rectum and vagina, where a spasmodic contractile pain is felt, or with the pain in the coccyx, there is a pain in the small of the back. The pain is liable to be worse in the night. The disease is generally very obstinate, and unless the neurasthenic basis is recognized, attempts at cure will be of little avail.

Treatment should in the first place be general and hygienic. Removal of the bone has often been suggested, and frequently performed. From this operation have resulted cure and failure in such equal proportion that I feel it to be largely psychic in its effects. I feel therefore that in the vast majority of cases, where no definite and causative traumatic condition can be demonstrated, operation should not be advised. The repeated application of moist heat for periods of two or three hours at a time, by the thermopile or the hot-pack, will give temporary relief, and be an aid to cure.

Faradic electricity has been advised, but I have never felt that it was of much benefit. If the pain is badly borne, and is severe, it may be alleviated for a time by the use of suppositories of opium or cocaine. Without neglecting hygiene or adjuvants, we may find less need of them if we select internal remedies with care. The principal drugs are: Aloes, Antimonium tart., Arnica, Belladonna, Cantharis, Carbo animalis, Causticum, Cicuta, Cistus Can., Fluoric acid, Kali bich., Kreosote, Lachesis, Magnesia phosph., Mercurius, Nux mosch., Paris quad., Petroleum, Rhus tox. and Ruta graveolens.

Meralgia paresthetica is a neuralgia of the femero-cutaneous nerve. It is not so much a pain as a paresthesia. It is probably caused by strains upon the nerve as it lies in the psoas muscle, or as it passes through the fascia lata, in a person suffering from the intoxication, infection, or anemia which lies at the base of all neuralgias. The symptoms are a burning pain in the nerve, rather annoying than distressing, which comes on spontaneously, or after sitting for a long time. Some patients find that it results from long standing or walking. The pain is most certainly relieved by bending the thigh upon the trunk, and thus relieving the strain upon the nerve. Added to this pain is the paresthesia, which gives the disease its name. It is a prickling numbness, which sometimes becomes an intense burning, and at the same time pain and thermic perception are reduced. At all times tactile perception is reduced, so that when the skin is touched it is perceived as pressure rather than touch. The treatment is one adapted to the general condition, and locally faradic brushing is advisable. Internally Aconite, Bovista, Cajuput, and Rhus tox.

Melalgia or Erythromelalgia is a somewhat similar form, in which claudication of the arteries of the leg is added to the neuralgic symptoms. There is in these cases, in addition to the paresthesias, a deep cyanosis of the lower leg, and severe pains in the calf, appearing very soon if the patient walks and stands, and very speedily relieved if the patient sits down, and at the same time elevates the feet. In this case the paresthesia may be very marked. In the one case which I followed for a long time there were exquisitely painful spots, real anesthesia dolorosa. According to Charcot the pains are caused by the contraction and obliteration of arteries. Erb attributes the disease to tobacco, as one-half his cases were heavy smokers. In the case referred to, the patient was a female who never used tobacco; numerous sections were excised, and most skillfully and exhaustively examined, and no suggestive change could be demonstrated. She was apparently cured by continuously sweating the limb by covering it with rubber tissue.

Metarsalgia (Morton's Painful Toe). This is a chronic painful sensation in the fourth metatarsal articulation, and it sometimes affects also the second or third. It arises from various causes. In some cases it is the evident result of articular bony changes, while in others we find a partial luxation of the metatarso-phalangeal joint. The larger number of cases reveal no such changes, and are supposed to be due to incipient flat-foot, or to the pressure of too tight or ill-fitting shoes, resulting in a pressure neuritis or neuralgia of the digital branches of the plantar nerves. Some cases seem to be purely hysterical.

The initial step should be an X-Ray of the bones, and whatever surgical procedure is thus indicated should be carried out. Exsection of the end of either the first or second metatarsal bone will always give relief. If it is not a surgical case, a wide and well-fitting shoe should be worn, and excavating the sole under the offending joint will at times give miraculous relief, when the widest and loosest shoe has not abated the pain in the least. Collins advises a shoe with a high heel, straight sole on the inner side, and a wide toe. Sometimes neurectomy is advisable. I have seen little benefit from electricity.

Remedies are Arnica, Cannabis Indica, Hypericum, Rhus tox., Ruta, and Sabadilla, according to O'Connor.

RAYNAUD'S DISEASE.

LOCAL ASPHYXIA, SYMMETRICAL ANGIONEUROTIC GANGRENE.

DEFINITION. This is an affection of the peripheral vascular system, which manifests itself in a spasmodic and recurrent contraction of the arterioles. While it is a general condition, it is most evident in the extremities. It may occur independently, or as a feature of other diseases, such as arteriosclerosis, hysteria, tabes dorsalis, syringomyelia, tumors of the spinal cord and spinal roots, epilepsy, exophthalmic goitre, scleroderma, neurasthenia, myelitis, and insanity, especially with acute mania.

SYMPTOMS OF A TYPICAL CASE. A poorly nourished, or neurotic person, by or before the thirtieth year, begins to have excruciating pains in the arms, or in the lower limbs. A numbness comes in two or three fingers or toes. They become bloodless, waxy, and shrunken, or blue and mottled; they are anesthetic, and if pricked do not show the injury by hemorrhage. After this condition has recurred a number of times during a few months, the parts become gangrenous, and finally undergo mummification. The condition is apt to be symmetrical, and if the feet have at first been affected, the hands are attacked later to a somewhat similar degree. The disease may stop at any point, or recede at any stage.

Differential Diagnosis. This must be from Chlorosis, Frost-bite, Peripheral Neuritis, Senile Gangrene, Gangrene from Embolism, Phlebitic Gangrene, Diabetic Gangrene, Ergotism, Erythromelalgia, Leprosy, and Ainhum.

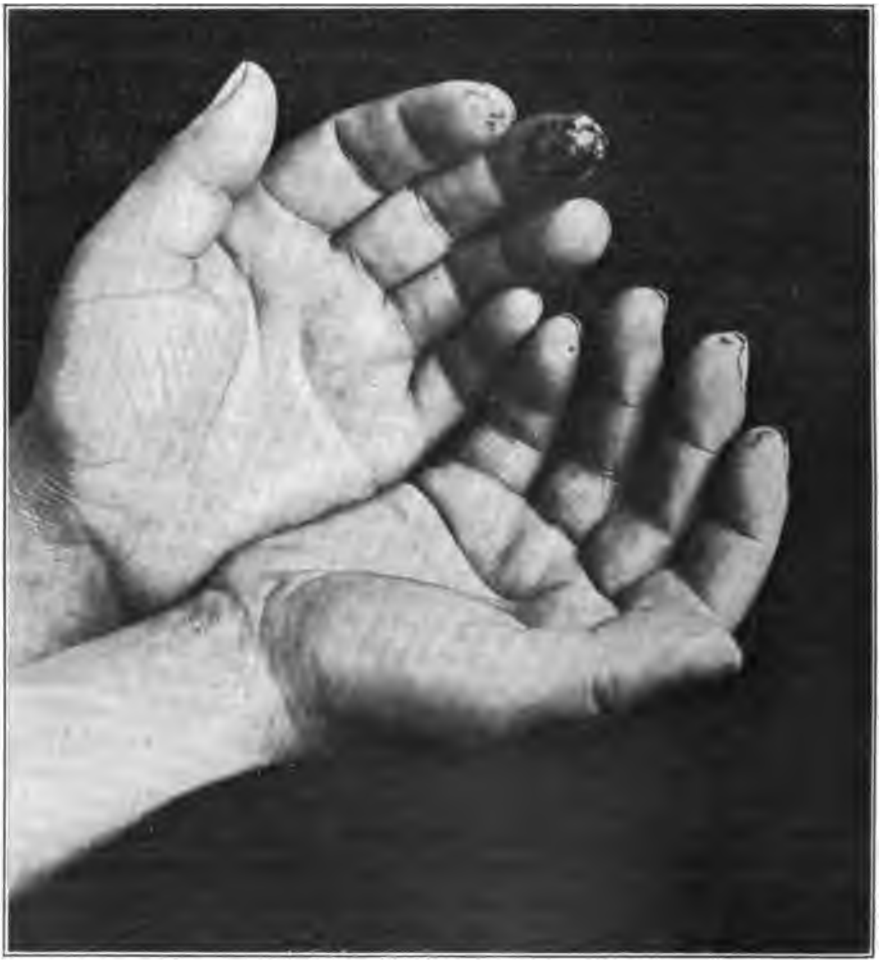


Figure 45. Raynaud's Disease.
Note especially the pointing of the figures, and incipient necrosis at the tip of each. (Howard Fox.)

Age. Most often between fifteen and thirty, but has been seen in nursing infants, and in old men.

Sex. The cases are females in the proportion of three to one, but those occurring in old age are generally masculine.

ETIOLOGY. Miuro says that there is heredity in 8%. All cases present evidences of a neuropathic constitution, and Stewart found in two cases an extreme vaso-motor reaction in both the affected and the normal extremities. This may be an indication of the truth of the hypothesis that the local lesions are due to an intense spasmodic vaso-constriction, with a resulting starvation of the tissues. On this neuropathic basis, emotional or physical strain, prolonged lactation, anemia, suppressed menstruation, exposure to cold, especially occupations exposing one to cold, or trauma, seem to have acted as exciting causes. Most cases are of foreign birth. A few cases have been attributed to morphine, or chloral intoxication. It has been known to follow the infections, and Bosanyi (and Lustgarten also) says that Raynaud's Disease may safely be regarded as one of the manifestations of hereditary syphilis, and 7% of the cases are associated with scleroderma. The most common causative condition has been a congenitally small aorta.

PATHOLOGY. The arteries and veins have shown no changes which indicate that primary changes in them are the cause of the disease, but from some influence an obliterating endarteritis is the final condition in most cases. It has therefore been ascribed to some perversion of the nervous supply to the vessels, which has produced long-lasting spasm. The operative lesion has therefore been localized by some in the sympathetic system, as the result of a primary disease of the spinal cord, which left the sympathetic system without adequate control. This was Raynaud's theory, and was based upon the fact that cases were helped by galvanization of the spinal cord. Weiss believes that the veins, rather than the arteries, are the seat of spasm, and at the same time regards the disease as trophic, and that vascular spasm is not the cause. Johannsen, Potein, Rollesten and Sachs have reported Raynaud's Disease and erythromelalgia in the same patient, and Voorhees therefore contends that they are due to undue contraction on the one hand, or on the other of dilatation of the terminal arterioles. The neuritis occasionally found is secondary.

SYMPTOMATOLOGY. Raynaud divided the symptoms into three stages, viz.: local syncope, local asphyxia, and local death. Normally the first symptom is bloodlessness of two or three of the fingers, and less often of the toes, preceded by hyperesthesia, which is followed by anesthesia, and an absence of hemorrhage from the prick of a sharp instrument. This condition may last from a few minutes up to several hours, and may be associated with chilliness, but no fever in any stage, nausea, and a feeling of general illness. This stage may not develope into the second, but may pass away, only to recur the next day, or in a few hours. After this stage the parts experience a reaction, and become red and puffed, instead of shrunken and cold, and with a stinging pain. After this the normal appearance is regained. Regression may occur at this stage. Several days may intervene between the attacks, but the interval may be marked by an intense burning pain which often precedes the initial attack. After several repetitions of the initial stage known as "Dead Fingers," or this initial attack may develope into it, the affected digits, after a cyanotic stage of an hour or two, become blue-black. This is the second stage of local syncope, or regional cyanosis, which may be present in one extremity, while another is in the first stage of simple coldness with a waxy-white color. The more common evolution of the disease is to find the toes upon both feet in relatively the same condition, and after they have suffered a loss of the terminal phalanges by mummification, to have the process manifest itself for the first time in the fingers, or vice versa. Riggs says that these first two divisions of symptoms are more properly Raynaud's phenomena; if the third stage is added, we have Raynaud's Disease. After the second stage has been reached several times, and been followed by regression into the period of puffy redness, we find that the condition is progressive, and that small blebs and bullæ may form, and slight hemorrhages may occur upon the pads at the extremities of the affected digits. These blebs are elevated, and ulceration follows, and then the parts become the seat of a dry gangrene. A line of demarcation forms, and the slough is thrown off in a few months. Suppuration is always slight. This does not necessarily involve a great amount of tissue, and commonly the patient emerges from the disease with the loss of the terminal phalanges on one or two toes or fingers, but sometimes it will recur, until, as in a case of the author's, successive amputations have been carried as high as the knee. This degree of severity in one extremity does not imply that the other members will be as severely, or as extensively involved. Bilateral attack and symmetry are almost the certain rule in the extremities, but while in addition to the extremities it may attack the inner aspect of the calves, the malleoli, the buttocks, the abdomen, the cheeks, and the tip of the nose, or ears, or may cause lividity of the breasts, it does not, in those localities, often produce gangrene. The genitals and the tongue are rarely attacked. As the process goes on to the more advanced stages, the pain becomes constant and excessive. I have known a patient to keep the feet protruded from a window in the coldest weather to seek relief from the distress. The general symptoms are depression and gastric disturbances in the prodromal stage. During the attack, sensation may be lowered in the affected parts, and movements become clumsy, on account of swelling in the soft parts, and rigidity in the tissues, muscles, and skin, and the patient may not be able to walk. Trophic disturbances may be discovered in skin, nails, and muscles, and the bones may show erosion. The superficial temperature of the affected parts sinks far below that of the rest of the body. Impairment of sight, hearing, and tinnitus have been noticed. Owing to the spasm of the coronary arteries, collapse sometimes occurs, and since the arteries in the kidneys are subject to the same influence, we find occasionally albuminuria, and glycosuria; hemoglobinuria has also been seen. Oculo-pupillary derangements, and paralysis of the iris, are sometimes present, owing to the action of the sympathetic, but aphasia is rare.

COURSE AND PROGNOSIS. These attacks may be slight, and recurrent through many years, whenever there has been an emotional strain, or a gastric derangement. The ordinary case lasts four months, and terminates in a mutilation of far less extent than the gangrenous area would indicate. A whole foot may become gangrenous, and yet the actual loss may be confined to the two terminal phalanges of one or two toes. In infants the gangrene may come on so rapidly, and be so extensive, as to cause death, and in the aged a like danger lurks in the probability of a resulting uremia.

DIAGNOSIS. The diagnosis rests upon the presence of local asphyxia, of all degrees up to gangrene, which has developed slowly with many recessions, and is symmetrical. There has been a neuropathic constitution, and the definite condition has been excited by some mental or physical strain. It is explained by no primary disease of the heart or blood vessels, and the presence of hemoglobinuria, or uremia, will add to the probability that it is Raynaud's Disease. The relative youth of the patient, and the peculiarity of the sensory disturbances are characteristic.

DIFFERENTIAL DIAGNOSIS. Chlorosis is a general, and not a local manifestation, and it is marked by the absence of sensory disturbances, and particularly by the absence of any intense degree of pain.

Frost-bite is similar in all its immediate manifestations, but the history is too definite to allow an error to be made.

Peripheral Neuritis is symmetrical, and has sensory manifestations of all grades, but they are not paroxysmal, and the nerve-trunks are sensitive. The pains and paresthesias are developed along the areas of distribution of definite nerves, while those of Raynaud's Disease are diffuse. Peripheral neuritis never eventuates in gangrene.

Senile Gangrene is a disease of old age, and is dependent upon recognizable changes in the vascular system. It is not paroxysmal, and while Raynaud's Disease confines its final destruction to a few phalanges, senile gangrene involves the loss of a whole toe, and in many cases the whole foot. In case Raynaud's Disease does involve a greater area, it almost always does so by *initial attack,* while senile gangrene tends to spread insidiously to any extent. There are rare exceptions to this general statement in the case of both diseases.

Gangrene from Embolism or Phlebitis is not paroxysmal, but has a definite localization from the first. These cases are not symmetrical, and have an ascertainable origin in valvular lesions, or cardiac thrombi in the embolic cases, while phlebitis leads to a moist form of gangrene.

Diabetic Gangrene has a history of glycosuria. Failing that knowledge, the case presents inflammatory symptoms, and a rapid, and not a paroxysmal course, or it has followed a traumatism, or there have been notable trophic disturbances of a nervous origin.

Ergotism. This has similar symptoms, but in cases of poisoning by ergot the gastro-intestinal disturbances have been of a very marked character.

Erythromelalgia gives rise to similar symptoms in the first two stages, but not often to the final one of gangrene, and it is commonly relieved by cold applications, which are not palliative of the pains of Raynaud's Disease, and is diagnostically relieved by the elevation of the affected part.

Leprosy. The specific alterations of leprosy are lacking, and in leprosy several fingers are attacked simultaneously, and fall off without pain.

Ainhum affects the toes more often than the fingers, is practically painless, and the slough is not preceded by blebs, or bullæ.

TREATMENT. No very definite plan of treatment has ever been developed, except upon very broad lines, all agreeing that the person should be well fed, and carefully guarded from cold. This avoidance of chill is particularly to be emphasized in respect to the parts affected, and these should be kept warmly wrapped. If it is within the means of the patient a warm climate should be selected. While food should be made the subject of special attention, it has been pointed out by one author that the use of stimulants is of doubtful expediency in the case of a neurotic person, since they so easily develope a reliance upon them. Lukewarm baths to the parts have many advocates, and the subject of massage is treated with a large divergence of opinion. Some say mild massage of the parts, others as strongly affirm that it is harmful, but that general massage of the neighboring parts is beneficial. It is said that one of the diagnostic points between this disease and erythromelalgia is that that is relieved by cold applications, while Raynaud's Disease is made worse by them; yet this is not invariably true, for as I have mentioned, a patient of mine was relieved by cold, and afterwards seemed to have shortened the attack by immersing the extremities alternately in very hot and very cold water, for one minute in each. If the parts are exceedingly painful a 50% solution of Menthol in alcohol may be applied, cotton wrapped over this, and a final covering of oiled silk.

Another method of controlling the intense pain is by applying a tourniquet to the limbs, or an Esmarch's bandage. When this is taken off the parts fill with blood, and the vascular spasm subsides. I believe that swathing the affected parts for days together with rubber tissue might be beneficial, by increasing the capillary circulation. Still another method of producing relief from pain is by applying the positive galvanic electrode to the parts, with the negative upon the spine. The affected part may be placed in a bowl of water, in which the positive has also been placed. The static spray has also been directed upon the affected parts, for the relief of pain. With the idea that either the spinal cord directly, or through its influence upon the sympathetic system, has been the cause of the disease, spinal galvanization has met with much favor, with the negative stationary upon the sacrum, while the positive is moved up and down the whole length of the spine, with a current strength of about fifteen milliamperes, for a period of not more than five minutes, care being taken that the current is not interrupted. General static treatment also seems to produce good results in some cases. The line of demarcation should be awaited before operation, as much more tissue may be saved than appears probable from the amount of apparent gangrene.

THERAPEUTICS. Secale cornutum in a somewhat low potency is probably the best remedy that we have to offer for the disease. The peculiarity in its symptomatology is the fact that it is one of the remedies affecting the tip of the nose. In the course of the process toward the developement of gangrene marked by the production of blebs and vesicles Arsenicum, Lachesis, and Crotalus should be useful. Glonoin has been advised, but it must have been on absolutely physiological, and not homeopathic grounds. There is a report by Halbert of a cure by Ferrum phos. 6x.

PHYSIOLOGICAL REMEDIES. These have generally been confined to measures to relieve pain, and the use of tonics. In addition to its tonic value, quinine has been advised if the patient falls under the suspicion of previous malarial infection. Nitrite of amyl, and nitroglycerin have been employed to overcome the arterial spasm, and thyroid extract is extolled by Short.

Salvarsan would naturally be considered, if evidences of syphilis are present.

DISEASES OF THE CRANIAL NERVES.

Structurally the cranial are similar to the spinal nerves, except that some of them are purely motor, and some of special sense only, while a part only conform to the spinal type in being both sensory and motor. The 1st, 2d, and 8th are nerves of special sense, and the first is actually a prolongation of the brain. The 3d, 4th, 6th, 7th, and 12th are purely motor, and arise from nuclei which are remnants of the anterior horns which have been cut off by the decussation of the pyramidal tracts. The 9th, 10th, and 11th are mixed nerves. Their motor parts arise from cell-masses lying in the central part of the medulla, and are upward prolongations of the vesicular column of Lockhart Clarke (as it is called lower down in the cord), and are visceral nerves. The sensory parts of these nerves are all grouped into one mass constituting the sensory portion of the 5th nerve. All sensory fibres originate in ganglia which are the analogues of the posterior root-ganglia of the spinal nerves.

Cranial nerves are not subject to functional diseases, for it has been proved that such diseases are localized in the nuclei, or in the brain, since cutting the nerve trunk, in cases of neuralgia, is not at all certain to relieve the pain formerly experienced in the distribution of that nerve. These nuclei are as liable to infection as are cell-aggregates in other parts. These nerves are means of transmission of impulses only. They, unlike spinal nerves, are very prone to be affected by organic disease. The interstitial structures are susceptible to all the diseases which affect similar structures in other parts of the body, and the fibres are just as liable to be affected by injuries and intoxicants. Their peculiarity of situation rather invites the occurrence of such conditions, and aggravates their effects, since these nerves are distributed to exposed portions of the body, they lie on hard bone in many instances, and they pass through bony canals which become points of painful constriction in the event of a swelling of the nerve-trunk, and the canals themselves are sometimes inflamed. The diagnosis between central and peripheral palsies is as essential as in the case of spinal nerves. A peripheral lesion abolishes function in the whole territory of distribution of the nerve, while a lesion of the nucleus may abolish the motor or sensory function solely in the case of a mixed nerve. When the question is between a lesion of the nerve or its nucleus on the one side, or a lesion in the tracts above the nucleus or the cortical representation on the other, the same reasoning applies as in the upper and lower neuron palsies in the case of spinal nerves. Upper neuron palsies, in this case called central, produce weakness of the muscles, with spasticity and increased reflexes, and no electrical changes beyond a degree of diminution of response to both currents. Peripheral lesions produce a paralysis in place of a weakness, which is flaccid; the reflexes are abolished or greatly diminished, and the muscles atrophy; there is electrically the reaction of degeneration; we find anesthesia or some other variety of sensory change, and nutrition of the parts innervated is impaired.

In the description of the diseases of the various nerves the adjuvant treatment will be separately described under each heading, but the therapeutics will be grouped at the end of the article.

Olfactory (1st) Nerve. This is actually a forward prolongation of the ventricle of the brain, and, in the embryo, exists as a hollow tube. This rod-like mass runs along the under side of the frontal lobe, and ends in a bulb lying on the cribriform-plate of the ethmoid bone. This plate has about twenty perforations, through which drop prolongations of the nerve which are distributed to the Schneiderian membrane, developing bipolar cells as they descend. In the ganglionic bulb is a layer of cells analogous

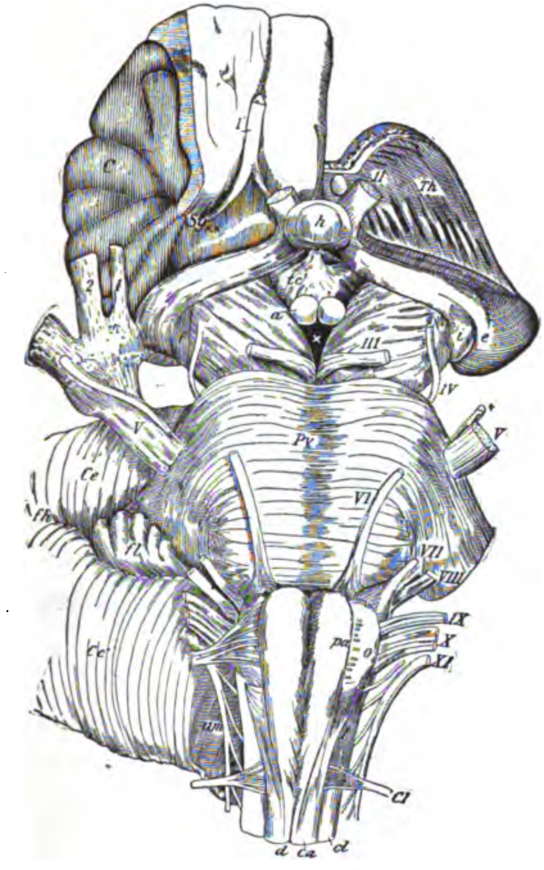


Figure 46. Brain Stem.

The base of the brain, and the crura, pons, and medulla, showing the superficial origin of the cranial nerves. I to XII, the cranial nerves; Th. optic thalamus; h, pituitary body; tc, tuber cinereum; a, corpora albicantia; P, pes pedunculi; i. interior, and e, exterior geniculate body; PV, pons variolii; pa, anterior pyramid of medulla; o, olive; d, decussation of anterior pyramids; ca, anterior column of spinal cord; cl, lateral column of spinal cord; Ce, cerebellum; fl, flocculus of cerebellum (Allen Thompson). (Starr. Courtesy of Lea & Febiger.)

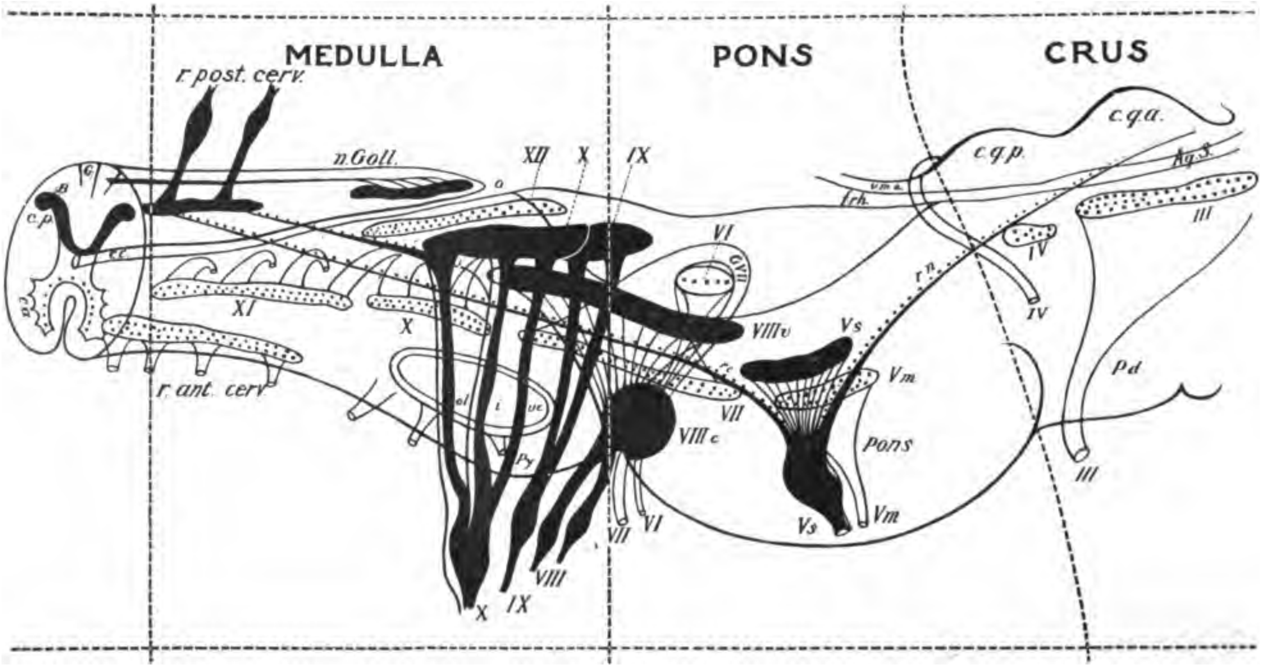


Figure 47. Deep Origin of all of the Cranial Nerves Except the Olfactory and Optic.
Motor nerves, and parts of nerves, are in outline, sensory in black.

to those found in the cell-nuclei of the cord, while the aggregation of bipolar cells represents the posterior root-ganglion of a spinal-nerve, and the peripheral fibres from these cells are the analogues of the sensory nerves. The cortical path of these fibres is very complicated, and in some parts in doubt, but is it not incorrect to say that they run first to the uncinate gyrus (which is the termination of the curved end of the callosal gyrus, which has turned over the forward end of the corpus callosum, and running backward lies on the temporal lobe just under the crus), of the *same* side of the brain, and thence, by way of the anterior commissure, proceed to the optic thalamus of the *opposite* side, and thence to the cortex in the parietal region by a tract in the posterior third of the posterior limb of the internal capsule. Smell is effected by odors acting chemically upon the sensory terminals distributed to the Schneiderian membrane. Loss of smell (anosmia), diminution of smell (hyposmia), or perversion (parosmia), may all be produced by injuries of the anterior cranial fossa, or severe intracranial pressure may produce atrophy of the bulbs, and consequent loss of smell, or tabes dorsalis may cause atrophy of the nuclei. The same result may ensue peripherally from the presence of polypi or catarrhal affections of the membrane. As a clinical fact it is well to remember that perversions of smell, while found infrequently in degenerations and growths of the cortical nasal centers, are more often indications of insanity. In testing a patient for aberrations of smell, one nostril must be closed while the other is being tested, and the mouth also, since many odors are as readily appreciated by the palate. Use alternately the pleasant and the unpleasant, as cologne, and then asafetida.

OPTIC (2D) NERVE. The structure of the optic nerve can best be understood by tracing it from its true, rather than from its apparent origin. At the back of the central part of the eyeball lies a layer of bipolar ganglion-cells like those on the Schneiderian membrane. This layer is the representative here of a ganglion on the posterior root of a spinal nerve. Each bipolar cell sends out a terminal fibre toward the center of the eyeball which is distributed on the retina, by a brush-like terminal, to an association with the brush-like terminal of either a rod or a cone. In a human retina are 130,000,000 rods, and 3,360,000 cones. This layer of rods and cones has resting upon its outer aspect, i. e., toward the front of the eyeball, a granular layer of pigment which is chemically sensitive to light. When light is admitted to the eye the granules group themselves about the tips of the rods and cones, and by this alteration in their position give to the observer the sensory stimulus which he has learned to appreciate as light. Toward the brain, or centripetally, run the opposite axons from these bipolar cells in the bundles which are known as the optic nerves. From each eye run from 450,000 to 500,000 fibres divided into centrifugal and centripetal fibres, which may be termed the axis-cylinder processes of the visual cells, while the parts extending into the retina, or the centrifugal fibres may be classed as dendrites. The great number of these fibres explains why a considerable degree of optic atrophy may exist, and yet the patient be unaware of its presence. The primary optic centres are the external geniculate body, the anterior collicus (or eminence of the corpus quadrigeminus), and the pulvinar of the optic thalamus. From these bodies new fibres pass to the visual cortex of the brain. When an adult is lacking a visual system, there is to be found no optic nerve, and no external geniculate body, but the eminence of the corpus quadrigeminus (anterior collicus), and the pulvinar are well formed. The optic tract is formed chiefly by fibres which terminate in the primary visual centers; von Monakow says that eighty per cent. terminate in the external geniculate body, and that all the visual fibres terminate in the primary visual centers. According to Edinger, the tracts contain some fibres which originate in the cerebral cortex. Von Gudden shows that the optic nerve contains both thick and thin fibres, and von Monakow says that the thick ones pass almost entirely to the anterior colliculi, and the corpora quadrigemina, and are not concerned with vision, but solely with the pupillary reflex. The arrangement of these fibres is in two bundles, an inner and an outer, and the combined bundles from the two eyes cross just in front of the anterior perforated space in what is termed the chiasm. As they come to the chiasm, the outer one, from the temporal half of the eye, continues directly backward, and enters the external geniculate body of the same side of the brain, thence to the anterior (called also the superior) corpus quadrigeminus, thence to the pulvinar of the optic thalamus of the same side of the brain, thence in a compact bundle through the posterior third of the internal capsule, and then, fanning out into what is known as the radiation of Gratiolet, is distributed to the cuneus of the same side, which lies on the inferior surface of the occipital lobe. The inner bundle crosses in the chiasm, but its further course is the same, though it goes to the nuclei of the opposite side of the brain. Henschen thought that he found that the upper part of the retina is represented in the upper part of the external geniculate body, and in the upper lip of the calcarine fissure; that the lower part of the retina is represented in the lower part of the external geniculate body, and the lower lip of the calcarine fissure, and the horizontal zone of the retina in the base of the calcarine fissure; from which it follows that fibres from the definite portions of the retina preserve their relative positions in the optic nerve and optic tract. Beevor and

Collier do not entirely confirm this in their necroscopy. Von Monakow says that hemianopsia is most likely to occur, and be persistent, perhaps only from a lesion in the calcarine fissure, though it may be possible in some cases from implication of the optic radiation, but he states that in every

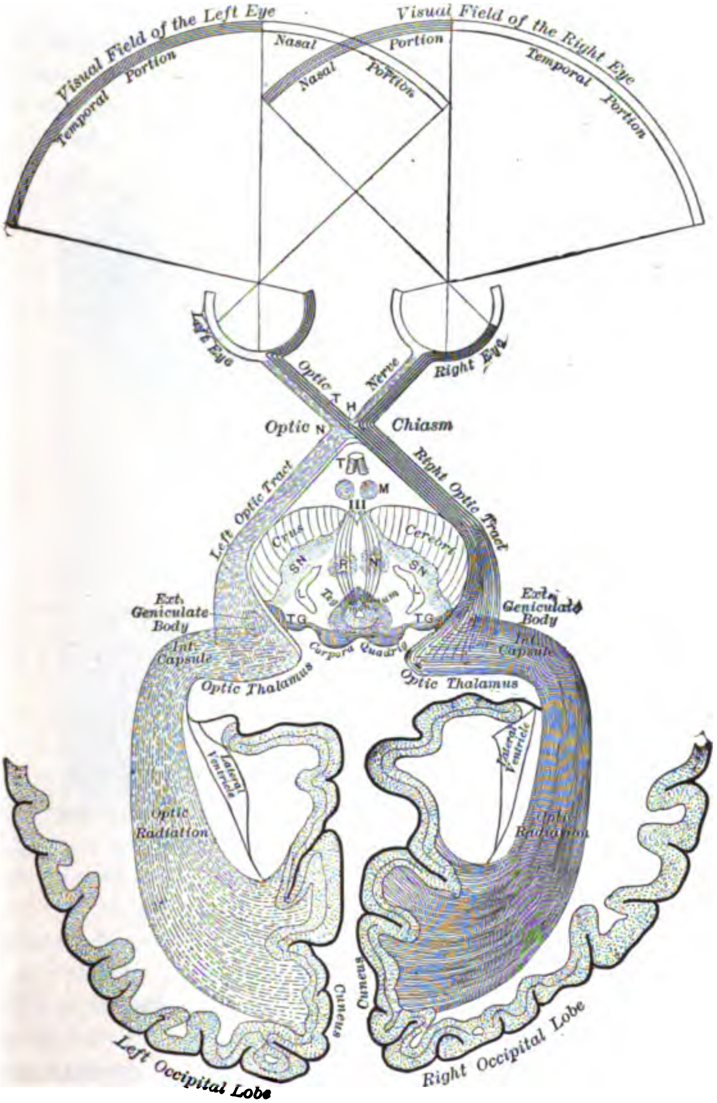
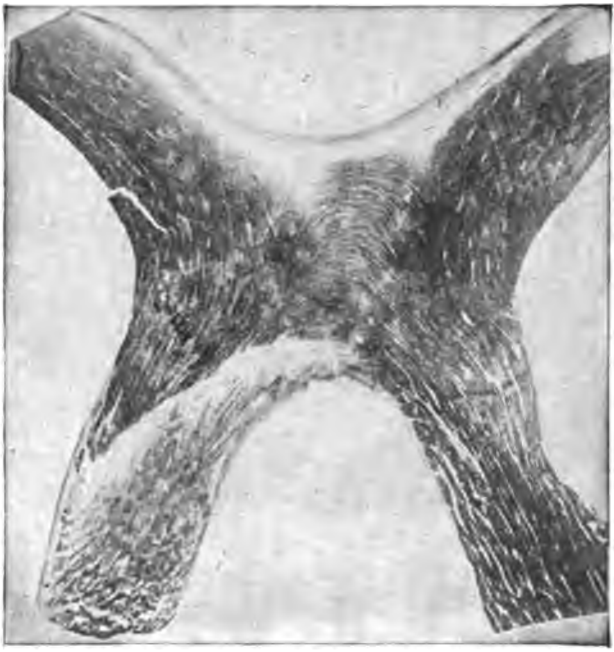


Figure 48. The Optic Radiation.

The visual tract. The result of a lesion anywhere between the optic chiasm and the cuneus is to produce homonymous hemianopsia. H, lesion at chiasm causing bilateral temporal hemianopsia; N, lesion at chiasm causing unilateral nasal hemianopsia; T, lesion at chiasm causing unilateral temporal hemianopsia; SN, substantia nigra of crus; L, lemniscus in crus; RN, red nucleus; III, third nerves. (Starr. Courtesy of Lea & Febiger.)

persistent cortical hemianopsia there is partial injury of some part of the occipital lobe, and, what is more important, a lesion of the optic radiation. There is a partial re-decussation of the fibres in the radiation of Gratiolet on their way from the internal capsule to the cunei. From these facts it is evident that complete blindness of one eye alone can only occur when the lesion is in front of the chiasm. These fibres are the centripetal ones, running from the retina to the cortex, and convey sensory impressions of sight. The other bundle is centrifugal and motor, and follows partially a similar route in a reverse direction. The point of divergence is at the anterior corpus quadrigeminus, where a portion leaves the main bundle, and running down the crus in the posterior longitudinal bundle is supposed to connect with the nuclei of the 3rd, 4th, and 6th nerves (motor oculi). There are also probably subordinate connections with other nuclei, but no one has been able to actually trace a physical connection between the optic nerve and the oculo-motor nucleus. It is generally supposed that it is by means of Meynert's bundle of fibres, passing in a curve below the aqueduct of Sylvius, although this may be a projection of the pupillary fibres. There



(Cut from Posey & Spiller, pg. 473, Fig. 3.) Courtesy of J. B. Lippincott Co.

FIGURE 49. Multiple Sclerosis. Lesion of the optic chiasm and one optic nerve. (Case of Dr. Spiller.

are also probably subordinate connections with other nuclei. This diversion creates a reflex arc by which we reflexly close the eye at the approach of danger to the eyeball. The macular fibres are the seat of the most acute visual perception, and those fibres are in a bundle by themselves, and most protected from injury, and seem most resistant to infection. In the chiasm are crossed fibres from the nasal half of each eye, uncrossed fibres from the nasal half of each eye, uncrossed fibres from the temporal half, and a crossing of the fibres from the two maculæ. The fibres to the external geniculate body are from the macula alone.

When the optic nerve is affected, the end showing in the middle of the retina, called the disc, becomes pale and atrophic. Optic atrophy may be primary or secondary. It is primary when the optic atrophy takes place without a preliminary inflammation of the nerve as a whole. There is no papillitis as a preliminary condition from which optic atrophy is to result. It is a primary degeneration of nerve-fibres resulting from disease of their nutrient cells in the nuclei. If secondary, it is almost always bilateral. The primary atrophy is generally from tabes, and is gray in color, disseminated sclerosis (where it is usually bi-temporal and white), general paresis, and, at times, from syphilis. The secondary form, after a preceding swelling of the nerve-head, is from intra-cranial tumors (a common cause), meningitis, renal diseases, lead poisoning, sclerosis, leukemia, pernicious anemia, diabetes mellitus. A very considerable degree of optic atrophy may be present without greatly impairing vision, on account of the resisting power of the macular bundle, but the legitimate result is blindness. Under such conditions we find that the disc is pale, there is concentric contraction of the visual fields and also of the color fields. In this contraction green goes first. The normal extent of the area in which the different colors can be perceived is white, blue, red, green. Hysteria may produce similar changes, and the diagnostic point is this: in hysteria the fundus looks normal, and the loss of vision is most apparent on the side of the body on which the loss of the sense of touch or pain is most evident. The loss of color perception is also eccentric in that the field for red or green may be larger than that for white, or there may be a symmetrical reversal (dyschromatopsia). It has been pointed out by Reichardt that disease of the optic nerve may cause complete blindness, and yet leave the reflexes of the iris intact. In compression, vision is affected more than the reflexes, and in acute inflammation there is improvement in the reaction of the iris before vision begins to return. In tabes the same author observed a case where a man had an atrophy of the left optic nerve, and had a normal pupillary reaction in that eye until four weeks before the loss of sight became complete, and at a later period even, the reaction could only be termed sluggish.

Keeping in mind the anatomical points just discussed, it will be evident that the lesion of one optic nerve between the eyeball and the chiasm is the only lesion that will cause complete blindness of one eye, and there will be blindness in one or both eyes, according to the depth to which a lesion penetrates the chiasm. Another reason for the usual escape of the macular bundle is the fact that it lies in the center of the chiasm, and a lesion must be deep to affect it. Central vision will thus escape in all partial lesions. Partial lesions of the tracts are apt to produce losses of half the visual field in both eyes, and this is called hemianopsia; if the same halves, i. e., right or left, are affected, it is called homonymus; if the blindness is of the right half of one eye, and the left half of the other, it is called heteronymus.

If the external portion of the chiasm is injured, all the fibres going to the temporal half of that eye will be involved, and such a loss will cause blindness to objects approaching the nasal side of that eye. We can get blindness of both temporal halves, seen on the nasal sides, only from a double lesion. This is heteronymous hemianopsia. Nasal hemianopsia has clinically never been found from a lesion behind the chiasm, and it has been said that a lesion of the chiasm does not explain its occurrence, since the fibres, crossed and uncrossed, are mixed to too great an extent in the external part of the chiasm. If a lesion occurs in the anterior part of the chiasm, the fibres coming from the nasal half of both eyes are affected, and blindness to objects coming from both temporal sides will result. This is heteronymous hemianopsia. If a lesion affects the optic tract after the decussation has taken place, we shall find a blindness of either the right or left halves of both eyes. This is homonymous hemianopsia. This term indicates it is the same half, either left or right, while heteronymous means the left or right of one eye and the opposite of the other. If a lesion involves the decussation, and also projects into one or the other limb, we shall find that there is temporal blindness of the eye opposite to the affected limb, and total blindness of the other eye. To sum up: we may say that

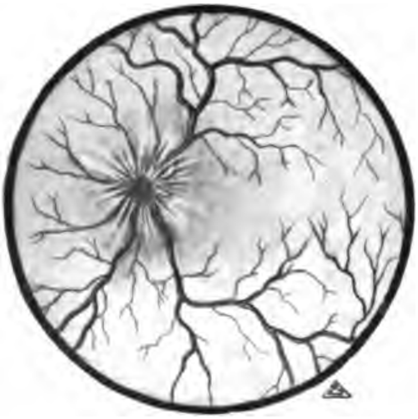


FIGURE 50. CHOKED DISC.

It will be noted that the clear outline of the disc is replaced by a very indefinite mass of opaque nerve-fibres.

from a lesion in front of the chiasm we shall get a blindness of one eye; a partial lesion of the chiasm produces hemianopsia of one or both eyes; complete destruction of the chiasm produces total blindness of both eyes; lesions back of the chiasm produce homonymous hemianopsias; and that in all hemianopsias central vision is undisturbed. Von Monakow says that hemianopsia as the result of a lesion confined to the anterior colliculus of the corpora quadrigemina has never been observed, and it is doubtful whether a lesion confined to the pulvinar, leaving the external geniculate body untouched, can produce a hemianopsia. The optic atrophy of spinal disease is gray, excepting in the case of multiple sclerosis, while that of primary disease of the nerve is white. The peculiarity in the case of disseminated sclerosis rests upon the fact that it attacks the anterior part of the chiasm. The mechanism of the pupillary reflex demands consideration.

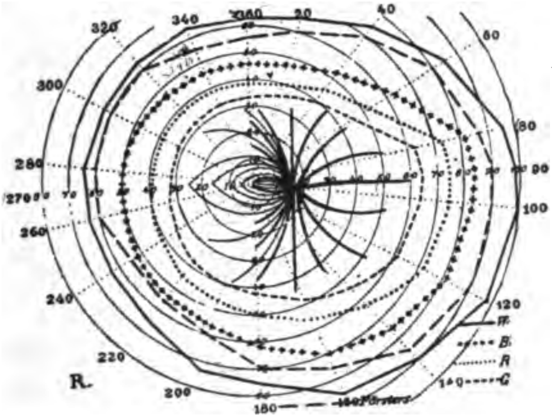
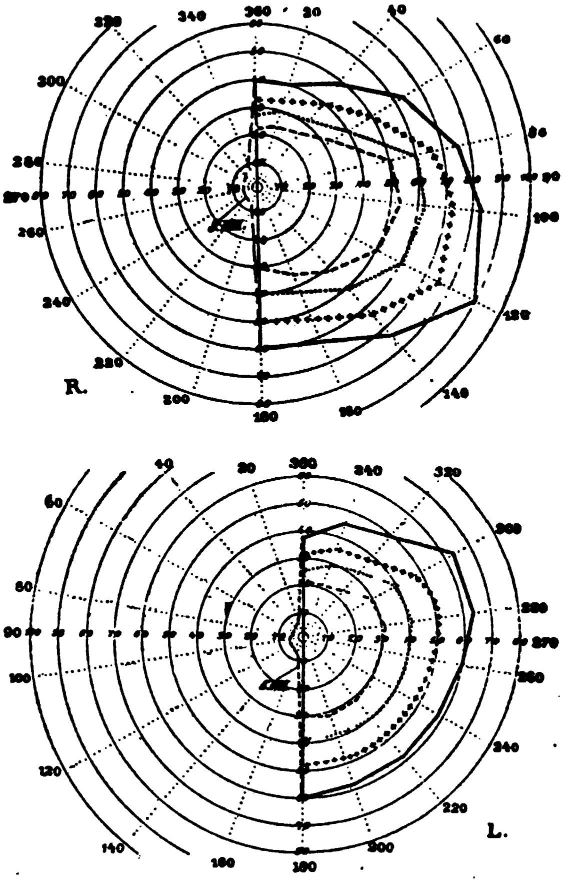


FIGURE 51. VISUAL FIELD OF THE RIGHT EYE.

The lines indicate the area for white; the stars that for blue; the fine dots that for red, and the large dots that for green. The black lines in the center represent the dispersion of the fibres of the optic nerve, which here have lost their medullary sheath. (From Baas.)

There are two sets of nerve-fibres, one contracting, while the other dilates the iris. Light falls on the retina stimulates the pupillary fibres which convey the impression to the nucleus whence a motor impulse is sent out by the path described. Reichardt assumes four pupillary centers in the cervical cord: (1) Budge's center, the so-called cilio-spinal center in the lower cervical cord (above the 4th cervical for rigidity); (2) a pupillo-dilator center in the upper part of the cervical cord; (3) an inhibition center for the light reflex; (4) a center for the dilatation of the pupil by irritation of the skin covering the cervical region in the fourth and fifth cervical segments. Bach and Meyer believe that in the upper part of the spinal cord, and the lowest part of the fourth ventricle, near the median line, there is an inhibition center for the light reflex, and an inhibitory center for pupillo-dilation. Irritation of this part always produces pupillary rigidity, and myosis (contraction of the iris), of the opposite eye. Reichardt, in cases of tabes and general paresis, found that in every case of pupillary rigidity there was a degeneration of the endogenous fibres, especially of the second and third cervical segments, in the area between the posterior commissure and the ventral part of the Column of Goll, the so-called Bechterew's intermediate zone.



FIGURES 52, 53. THE VISUAL FIELDS OF A CASE OF LEFT HOMONYMOUS HEMIANOPSIA.

The dividing lines between the seeing and non-seeing halves is seen to be perpendicular, but without curving round the centre or fixation point as it usually does. In this instance, however, the field for green is seen to extend beyond the centre. The solid line indicates white, the stars blue, the fine dots red, and thelarge dots green.

The treatment of these cases generally falls to the oculist, but the hemianopsias are to be treated according to the causal indications.

OCULO-MOTOR, OR COMBINED 3D, 4TH, AND 6TH NERVES. These three cell-groups lying under and in the floor of the aqueduct of Sylvius and of the fourth ventricle, are joined by association fibres into a perfect coordination. "The axons composing the 3rd nerve arise from a series of seven or eight groups of nerve cells. Enumerating them from before backward, the nuclei occur in the following order:

(1) The sphincter pupillæ.

(2) The tensor choroidæ (the accommodation nucleus).

(3) The convergence nucleus, a common nucleus for the conjoint action of the two internal recti muscles.

(4) The superior rectus.

(5) The inferior rectus.

(6) The levator palpebræ.

(7) The inferior oblique." Brubaker, pp. 579-580.

The 3rd (motor-oculi) nerve reaches the surface of the inner side of the crus cerebri at the upper surface of the pons, and goes through the outer side of the cavernous sinus, and thence enters the orbit by the sphenoidal fissure. It has upon it the ciliary ganglion (ophthalmic or lenticular), which lies at the back of the orbit between the external rectus and the optic nerve. This nerve supplies the ciliary muscle, sphincter of the iris, levator palpebræ, superior rectus, internal and inferior rectus, and the inferior oblique. Fourth (trochlear) nerve arises from a small nucleus in the floor of the aqueduct, and is peculiar in that it decussates with its fellow in the roof of the aqueduct just beneath the posterior corpora quadrigemina, and immediately after this makes its exit upon the dorsal surface of the crus, lying upon the superior peduncle of the cerebellum. It then curves about the crus, resting upon the upper border of the pons, passing into the cavernous sinus at its outer side, and enters the orbit by the sphenoidal fissure, and is distributed to the superior oblique. Sixth (patheticus) nerve has its nucleus in the floor of the 4th ventricle, quite a little below that of the 3d, and 4th, the connections of the three making possible the conjoined movements of the eyeball, but the 6th nucleus is the dominant one, and it receives first the impulses from the brain. It emerges just at the lower margin of the pons, and near its center, and enters the cavernous sinus at its center, lying just under the internal carotid artery, and enters the orbit by the sphenoidal fissure, and is distributed to the external rectus muscle.

Paralyses of these nerves arise from injuries to them anywhere in their course from their nuclei to their distributions. They may be injured by new-growths, exostoses, especially vulnerable to the effects of thrombosis of the cavernous sinus, chronic inflammatory conditions and tuberculosis, whether in the bones, meninges, or brain-substance. The exudates of syphilis or tuberculous meningitis may permeate their structure, or may compress them in continuity. In the orbit they are open to damage from hemorrhages, thrombosis, or new-growths, and there may be a parenchymatous degeneration from cold or alcoholism, or an interstitial inflammation may be a result. The infections are the most common causes of a degeneration of the fibres, and of these infections diphtheria is the most potent. This infection is the most common cause of a bilateral paralysis of the 6th, although the 3d may also be implicated. Disseminated sclerosis often produces a partial or complete paralysis of the oculo-motor group, and tabes, or general paralysis of the insane (general paresis), are the most frequent causes of pupillary rigidity. The 3rd nerve supplies all the orbital muscles except the superior oblique, and the external rectus, and in addition, the nucleus of this nerve, through communication with the roots of the facial, supplies the orbicularis palpebrarum, occipito-frontalis, and corrugator supercilii; but the fibres of the 3rd nerve to the internal rectus are derived from the 6th nerve nucleus of the opposite side.

Lesions may be either irritative or destructive. "Stimulation of the nerve near its exit from the encephalon is followed by contraction of the muscles to which it is distributed with the following results, viz.:

(1) Diminution in the size of the pupil.
(2) Accommodation of the eye for near vision.
(3) Elevation of the upper eyelid.
(4) Internal deviation and rotation upward and inward of the anterior pole of the eye, combined with a small amount of torsion toward the mesial line, due to preponderating action of the internal rectus and inferior oblique muscles.

Division of the nerve either experimentally or as a result of compression from a pathologic cause is followed by a relaxation of the muscles, with the following effects, viz.:

(1) Dilatation of the pupil, the iris responding neither to light, nor to efforts of accommodation.
(2) Loss of the accommodative power.
(3) Falling of the upper eyelid (ptosis).
(4) External deviation and rotation downward and outward of the anterior pole of the eyeball, combined with a small amount of torsion toward the mesial line due to the unopposed action of the external rectus, and the superior oblique muscles.
(5) Double vision or diplopia. The image of the eye of the paralyzed side is projected to the opposite side of the true image, and to the

upper part of the visual field. Owing to the slight medial torsion, the false image is inclined away from the true image.

(6) Immobility and slight protrusion of the eyeball." Brubaker, pp. 580-581.

It should be remembered that there is a form of migraine which is marked by a transient oculo-motor palsy, especially of the 3d nerve, which may occur at regular intervals, and be followed by complete recovery. Charcot thought it depended upon an irritation of the vaso-motor fibres. At times it is accompanied by a blunting of sensation in the distribution of the 5th nerve.

Paralyses of the 4th nerve are rare, and difficult of diagnosis. When paralysis does occur on account of the unopposed action of the inferior oblique muscle, the eyeball is turned upward and inward, with a slight torsion in the middle line, giving rise to a diplopia which is homonymous, the images being one above the other.

"Isolated paralysis of the 4th nerve without affection of the 3rd or 6th is an important symptom of tumor of the cerebellum, or of exudation on the under surface of the anterior lobe of the cerebellum" (Starr).

Paralysis of the 6th nerve is the most frequent of oculo-motor palsies, this nerve being especially affected by syphilis. The result is that the unopposed internal rectus turns the eyeball inward (internal strabismus) and this gives rise to a diplopia which is homonymous, while the images are on the same level and parallel.

TREATMENT. Syphilitic cases should be energetically treated. Other cases should be treated on the basis of their causation. If the eye continues to deviate after treatment use electricity. Apply a bipolar, faradic electrode over the conjunctiva after cocainization, at the insertion of the muscle. Use the very weakest current. Another method is to apply a galvanic current over the eyelid, or along the margins of the nose and the inner margin of the lid, with the negative sponge electrode, and place the positive on the nape of the neck.

FIFTH (TRIGEMINAL) NERVE. This is a mixed nerve, and the motor-root rises in a nucleus in the pons. The sensory roots are three in number, and actually have their origin in the Gasserian ganglion, which lies in the cavum Meckelii (a cavity formed in the dura on the upper surface of the petrous portion of the temporal bone). The roots of this run backward to the pons, whence one group ascends to the cells of the aqueduct (formerly termed the descending root), another descends into the substantia gelatinosa of the upper segments of the cord (formerly called the ascending root), and arborizes about these cells, and those of the nucleus cuneatus and gracilis on the anterior surface of the medulla. This mixed nerve appears at the surface of the pons at about its middle, and thence, going forward on the under surface of the tentorium, spreads out as it enters the Gasserian ganglion, while the motor part passes under it. On its emergence from the ganglion, the sensory roots are found to have undergone division into three parts, with the lower of which the motor-root unites. These divisions are the ophthalmic, superior maxillary, and inferior maxillary. The ophthalmic is about an inch long, and running through the outer wall of the cavernous sinus enters the orbit by the sphenoidal fissure. It is purely sensory, and at the entrance of the orbit gives off branches to the 3d, 4th, and 6th nerves. This branch of the 5th furnishes the sensory innervation to the skin on the front of the head, the conjunctiva, cornea, iris, mucous membrane of the frontal sinus, and a part of that of the nose, and probably sensory fibres to the tear-glands. The superior maxillary branch has a short intra-cranial course, in which it passes through the outer side of the wall of the cavernous sinus, emerges from the skull by the foramen rotundum, and enters the orbit by the spheno-maxillary fissure, and passes on to the floor of the orbit. It then, through the infra-orbital canal, passes to its termination in the face. It supplies the skin of the face between the eyes and the mouth, the mucous membrane of the upper jaw, the naso-lachrymal duct, the lower part of the nasal mucous membrane, and that of the gums of the upper jaw, the middle ear, the antrum of Highmore, sensory fibres to the upper teeth, and probably some of the taste fibres.

The inferior maxillary unites with the motor branch, and after a short course leaves the skull by the foramen ovale, and about a quarter of an inch beyond the skull breaks up into its branches. It innervates the skin of the lower jaw, external ear and temporal region of the scalp, the mucous membranes of the tongue, lower jaw, cheek, lower lip and teeth. It furnishes the motor fibres to the muscles of mastication, i. e., the masseter, temporal, internal and external pterygoids, and to the tensor tympani, stylo-hyoid and the anterior belly of the digastric. Upon the 5th nerve are four small ganglia, each having a motor, sensory and sympathetic root. The ophthalmic, ciliary or lenticular ganglion is 1-12th of an inch in diameter and is situated at the back of the orbit, and imparts sensory and trophic innervation to the eyeball, motion to the ciliary muscle and sphincter of the iris (motor fibres from the 3d), and sympathetic fibres to the dilator of the iris. The spheno-palatine or Meckel's ganglion is 1-5th of an inch in diameter, and lies in the spheno-palatine fossa. Its branches have a very wide distribution to the structures of the face and the sinuses. The submandibular ganglion is of the size of a pin's head, and is situated under the mylo-hyoid muscle, and sends branches to the glands under the tongue. The otic ganglion is 1-6th of an inch in diameter, and lies close to the foramen ovale, and sends, most essentially, fibres to the tensor palati and tensor tympani.

Primary disease, excepting neuralgia, is rare. A primary neuritis is unusual, and the nerve is rarely involved in a multiple neuritis, but is most often found to be diseased in a secondary way. Any tissue change or new growth in the posterior and middle fossæ of the brain, or tumors, or softenings in the pons and medulla, will injure the nerve in some of its nuclei of origin, or in continuity. If the softening is in the cord, the so-called ascending root only is compromised, and so the lesion will be wholly sensory, while if the lesion is in the pons, the motor root or nucleus will also be affected. In the cord it has been involved in syringomyelia. Tumors, gummata and exostoses at the base of the skull are apt to involve it, and also growths infringing on the cavernous sinus and the orbit. Operations about the jaw and orbit endanger it in some of its branches. Gummata sometimes affect the nerve directly. If the trunk of the nerve is affected, we shall naturally find motor, sensory, trophic, and secretory symptoms, but the sensory losses are not always as great as might be predicted, owing to the richness of anastomosis between sensory nerves in all situations. In such accidents the secretion of tears ceases, smell is diminished, as well as sensation in the teeth, cheeks, lips and gums. If we wish to test the sensory condition of the lips, we can place a piece of glass between them when the mouth is closed, and the glass will appear to be broken at the point where sensation is lost. Hyperesthesia is apt to occur first, anesthesia later, while photophobia is a common symptom. It has been said that taste is also lost in these cases, but this is not true, accurately speaking. Taste in the posterior one-third of the tongue is known to be from the 9th nerve, but in the anterior two-thirds from the corda tympani of the facial, but those fibres also actually come from the 9th. The dependence of the preservation of taste upon the integrity of the 5th nerve is clinically true, but it depends upon the following fact; the production of moisture on the mucous membranes depends upon the sympathetic branches in the 5th, and when that is paralyzed the surfaces are dry, and taste is blunted to abolition, or near to it.

The most evident result of trophic loss is herpes in the distribution of the nerve, and it will involve the eyeball if the ophthalmic branch is affected. We sometimes find partial blindness resulting from lesions of the branches of the 5th, and it is marked by a considerable degree of concentric contraction of the visual field, and some loss of acuteness of vision. It is principally found in diseases of the dental branches, especially those to the molar teeth. In the severe neuralgic cases an X-Ray examination of the teeth and alveolar processes should always be made, as it has been found that pathological processes may exist in this locality, and still escape the diagnostic methods of the dentist. It is the most fertile cause of neuralgias of the fifth nerve. If the cerebral centers for the nerve are diseased, we find severe trophic symptoms, such as ulceration of the cheeks and gums, and falling out of the teeth. This is principally caused by tabes. Paralysis of the motor branch involves the muscles of mastication, and, if bilateral, abolishes the reflex. In all peripheral affections there are reaction of degeneration, and more or less atrophy. In simple compression the symptoms are irritative, and if of the sensory branches, it may produce facial spasm, cough, vertigo, and other reflex phenomena.

TREATMENT. These paralyses must be treated as are palsies elsewhere, whether they are motor or sensory. If syphilitic they must have specific treatment, and the eyes should be carefully protected. For treatment of tic douloureux see article on Tic Douloureux under Neuralgias.

PROGRESSIVE OPHTHALMOPLEGIA, Polioencephalitis superior, is a disease which in acute, subacute, and chronic form involves to a variable degree the whole oculo-motor nuclear apparatus.

In its acute form we find cases where a person will lose oculo-motor efficiency after a period of unconsciousness of a few hours, or in the subacute form in a few days. The cause of these cases is very varied, and they have been seen to follow infections, and be the result of intoxications, have been seen after poisoning by lobster, and have been the result, after a confusional period of a few days, of a fall on the back of the head. Oppenheim states that the traumatic cases are the result of a hemorrhage near the aqueduct. In subacute cases there is no positive method of determining whether the lesion is a nuclear or a neuritic one.

The chronic condition is also termed progressive ophthalmoplegia, and progressive upper bulbar palsy. Von Graeffe first described this disease, which is a progressive invasion of the oculo-motor nuclei, although all may be involved simultaneously, which is essentially chronic in its course. In the course of some months, or perhaps years, all the nuclei may be involved upon both sides of the aqueduct. The disease has sufficient coherency in its grouping of symptoms to enable a division to be made into ophthalmoplegia interna, and externa, the sphincter of the iris and the muscles of accommodation being affected in the first group, and the external muscles of the eye in the second. Anatomical examination has shown, according to von Monakow, that nuclear disease could always be demonstrated in the internal form. After years, the sphincter of the iris and the levator palpebrarum superior may remain intact.

The etiology of the chronic form comprises the same intoxications and infections as in the acute form, except that alcohol and lead are very common causes, and that syphilis bulks more largely than was supposed before the use of the Wassermann tests. It often represents a congenital disease, or one acquired early on the basis of a hereditary familial disease. In a large percentage of the cases it is an early element in the symptom-complex of tabes dorsalis, progressive paralysis, combined system diseases of the cord, multiple sclerosis, progressive bulbar palsy (which is polioencephalitis *inferior* chronica), or progressive muscular atrophy. In other cases it is an upward extension of these diseases. It affects the sexes about equally, although some assert a masculine preponderance. It usually developes between the ages of fifteen and forty, but may attack an older person.

PATHOLOGY. The pathological changes are a chronic inflammatory or degenerative process in the floor of the 3rd ventricle and the aqueduct of Sylvius, which sometimes results in a thickening, which produces an atrophy of the ganglion cells, causing a degeneration of the whole nucleo-peripheral neuron. It may be a peripheral neuritis, and these are the stationary cases to be referred to below.

SYMPTOMATOLOGY. As a rule the affected person does not discover his condition for a long time, inasmuch as he may have perfect general health, although in rare cases an implication of the sensory root of the 5th nerve may give rise to facial anesthesia, or more probably paresthesia. He may notice that the drooping eyelids give him a sleepy expression, and he has to turn his face upward to see clearly. This is called "Hutchinson face." His vision is not disturbed, and there may be no diplopia. On examination, however, it will be found that the motility of the eye is almost abolished, and the excursions of the examiner's finger can be followed to a very limited extent, although as a rule the iris reacts to light and accommodation. There may be a slight squint leading to diplopia, but the progress of the disease is so slow that the patient accommodates himself to monocular vision. Diplopia is, however, fairly common.

PROGNOSIS. The disease may be years in evolution, and it may reach a stationary stage, which in a case of Struempell's lasted twenty-five years. If it is the upward extension of a progressive muscular atrophy it is fatal in two or three years.

DIAGNOSIS is from asthenic bulbar palsy (mysthenia gravis) and Tumor in the region of these nuclei. From asthenic bulbar palsy the diagnosis should be made by noting that any variety of function may be perfect for a very short period, to be followed perhaps by an easy fatigue in the palsy, while the symptoms, whatever they may be, in ophthalmoplegia, are constant. See asthenic bulbar palsy.) Tumor may counterfeit all the symptoms, but in a short time the general tumor symptoms will present themselves, and clear up the diagnosis.

TREATMENT. This must be causal in so far as remedies are concerned; efficient curative measures are doubtful, and the adjuvant is the same as when individual muscles are paralysed.

SEVENTH (FACIAL) NERVE arises from a nucleus in line with that of the 5th and the 9th, and it has a tortuous course in the pons, where it runs backward, and then upward, and then to the outside, and then forward on the outside of the nucleus of the 6th. Before it leaves the pons it is joined by the pars-intermedia, which is from the 9th, and embraces the fibres which are afterward to appear as the chorda-tympani. It emerges at the base of the pons at the lateral margin of the medulla, and enters the internal auditory meatus, and thence goes to the Fallopian canal, and emerges from the skull at the stylo-mastoid foramen, while in the Fallopian canal it gives off the chorda-tympani (taste), and a branch to the stapedius.

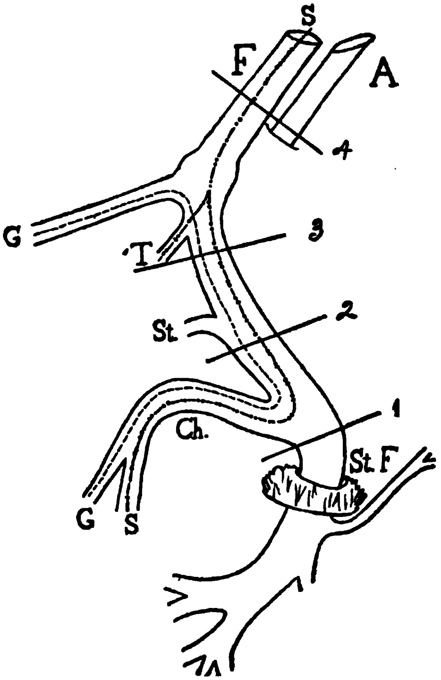


FIGURE 54. Diagram of facial nerve passing through the petron. F, Facial; A, auditory; St. F, stylo-mastoid foramen; Ch, chorda; S, S, salivary fibers; G, G, gustatory fibers; St, branch of stapedius muscle; T, tympanic branch. (Church & Peterson. Courtesy of W. B. Saunders Co.)

After leaving the stylo-mastoid foramen it passes into the parotid gland, and on its emergence divides and interlaces its branches to such an extent that this portion is termed the "pes anserinus." This nerve which is motor, barring the pars intermedia, supplies the buccinator (the tensor of the cheek), the superficial muscles of the face, of the scalp, external ear, nose, mouth, eyelids (except the levator palpebræ superioris), and the platysma, the muscles of the tympanum the levator palati azygos-uvulæ (through the large petrosal nerve), the stylohyoid, and the posterior belly of the digastric.

Facial paralysis, prosopoplegia, or Bell's is the most common of all the cranial-nerve palsies, and it is central if previous to the exit of the nerve from the pons, and peripheral at any point outside it. The chief causes seem to be cold and exposure, but probably these are only localizing factors to existing infections of which rheumatism is a common one. Facial palsy has come from ptomaine poisoning. The actual pathological change is generally a degeneration of the fibres. Clinically it follows middle-ear disease, caries of the petrous portion of the temporal bone, mastoiditis, tonsillitis, mumps, articular rheumatism, gout, diabetes mellitus, diphtheria, grippe and leukemia. Collier says that conditions primarily affecting the parotid gland are the almost universal cause. Traumatism at almost any point in its course or distribution has caused it, even the extraction of a tooth. A multiple neuritis may extend to the facial. It may be caused by forceps, and is then usually bilateral, and it may arise from atrophy of the nucleus, congenital in time, or from cortical degeneration, or lesion of the path from the cortex.

SYMPTOMATOLOGY. It is apt to come on suddenly in the night without warning, but may be preceded for some days by pain in some of the sensory nerves, in the ear, back of it, in the face or neck, or in the occipital branches. There may be fever, headache, or tinnitus. When we look at the face the lack of symmetry first strikes one's attention, since one side is smoothed out, and all expression is gone. The corners of the mouth droop. The patient cannot whistle, since the buccinator is paralyzed, the eye is wide open, the mouth also, and in chewing the food drops from the mouth, or accumulates between the teeth. Taste is absent from the anterior two-thirds of the tongue, although that organ can be protruded naturally. Saliva may be decreased from the affected side, and sometimes there are herpes zoster, and swelling of that side of the face. Muscular atrophy is common, there is the reaction of degeneration, and its degree is the basis for prognosis. Hearing is, at times, unusually acute.

Dana says "the 7th nerve has a sensory part, and its disorders cause otalgia of simple type, an otalgic tic douloureux, and a herpetic otalgia, due to lesions of the geniculate ganglion. When both motor and sensory fibres are involved, there results the 'Hunt syndrome' of facial palsy, otalgia, and herpes of the ear."

DIAGNOSIS. There is no difficulty in well-marked cases, but the localization of the lesion is important. It may be cortical, or in the nucleus, or peripheral. If it is cortical there is no reaction of degeneration, the upper part of the face is spared, and there is apt to be a paralysis of the extremities to some extent. If the lesion is in the pons, just after the fibres have decussated to go to the nuclei, there will be a crossed hemiplegia (face of one side, and extremities of the other), which is a mark common to all pontile paralyses. Reflexes are either normal or exaggerated. If it be nuclear, all branches are usually involved, but the upper face may here also be spared. Reaction of degeneration is present, and the reflexes are abolished. The 6th and the 8th nerves are also involved. If peripheral, we also have the question of a more exact localization, on account of the site at which to apply treatment if any local measures are employed. If it is attacked at the base of the skull, we shall find paralysis of the auditory and other cranial nerves, and the general cerebral symptoms of headache, vertigo and vomiting. If in the nerve-trunk internal to the chorda-tympani, taste and hearing will be affected, while if outside, all the facial muscles will be paralyzed, but these senses will be spared.

PROGNOSIS. In cases from rheumatism the severity will be moderate, from infections doubtful, and if from caries of bone only to be benefitted by surgery. The prognosis is based upon the extent of the reaction of degeneration. If after two weeks the reaction is normal, or only slightly disturbed, the case will recover speedily; it has a good prognosis, though the cure will be long delayed if a partial reaction of degeneration is found, but the prognosis is grave or hopeless if the reaction is complete.

TREATMENT. If the case is syphilitic treat specifically, while if rheumatic hot cloths should be applied to the region of the mastoid, and the parotid gland. Some strongly advocate leeches in the early stages. If it is in the canal, or from caries of bone, the relief must be from surgery. Electrical treatment can be used at all stages. At first stabile galvanism, and later labile, and, if neuritis is present, use only the mildest currents. Anode on sternum, and cathode to the nerve where it emerges from the stylomastoid foramen. No faradism at first in severe cases, and when it is used, employ very slow interruptions.

The facial type has been helped by connecting into the hypoglossal.

EIGHTH (AUDITORY) NERVE. This is a mixed nerve of hearing and equilibrium. It has three nuclei in the floor of the fourth ventricle, and emerges in a groove between the pons and the medulla. It then accompanies the facial into the internal auditory canal, and is divided into two parts, the smaller of which is the upper. This goes to the utricle, and the superior and external semi-circular canals, and subserves the function of equilibration. The lower and larger division goes to the organ of Corti in the cochlea, and sends off twigs to the saccule and the posterior semi-circular canal. This is the nerve of actual hearing. It connects with the pars-intermedia and the geniculate ganglion on the facial. This nerve is rarely the subject of isolated disease, but diseases of the middle ear often extend to its terminal branches in the labyrinth. Ménière's disease is supposed to be caused by a hemorrhage in this nerve. The causes of possible diseases of the cranial nerves are effective in the case of this nerve.

NINTH (GLOSSO-PHARYNGEAL) NERVE. The nucleus is near that of the 10th, in the floor of the fourth ventricle under the fovea inferior. It leaves the medulla at its upper part between the olive and the restiform body, and passes through the jugular foramen along with the 10th and 11th nerves. It is connected with two ganglia, the jugular and the petrous. It runs downward in company with the internal carotid artery and the jugular vein, and crossing the stylo-pharyngeus is distributed to the tongue, pharynx, middle ear and stylo-pharyngeus muscle. It connects with the 5th by the otic ganglion; with the 7th by the trunk and branches; with the 10th in the same way, and with the sympathetic. It is motor to the stylo-pharyngeus, and sensory to taste and touch. It is affected by the same conditions as the other nerves, but is peculiarly liable to suffer from injuries, inflammations, and thrombosis of the jugular vein. Symptoms of its involvement are anesthesia, and loss of taste in the posterior third of the tongue, and difficulty of swallowing.

TENTH (PNEUMOGASTRIC) NERVE. The roots are in the trigonum vagi in the floor of the fourth ventricle. The nucleus ambiguus is supposed to be the origin of its motor root. The sensory fibres are the axons of the root- and trunk-ganglia which lie in, and below, the jugular foramen, and are the petrous and jugular ganglia. These axons enter the medulla, and arborize about the cells of the so-called combined nucleus of the 9th and 10th, which is the homologue of the column of Clarke in the lower levels of the cord. The nerve now passes out between the olive and the restiform-body, runs upward to the jugular foramen, in which it unites with the accessory portion of the spinal-accessory nerve. It now passes down the neck, and is chiefly distributed to the viscera. It is the longest cranial nerve, since, after going to the pharynx, larynx, heart, lungs, esophagus and stomach, it still further extends to the liver and kidneys. Its functions are motor, sensory, excretory and also vaso-motor.

It suffers like all other cranial nerves, and a primary neuritis is rare. It is peculiarly liable to injury from growths and accidents at the base of the brain, and suffers in multiple neuritis, and from infections and intoxications. In total paralysis from these causes, coincident paralysis of the 9th, 10th and 12th occurs. It is usually implicated in diseases affecting the bulb, in tabes, multiple sclerosis, hysteria, and the neuroses from vascular changes in the jugular vein, and thrombosis of the transverse sinus. In its long course in the chest and abdomen it is liable to pressure and infection from contiguous inflammations.

SYMPTOMATOLOGY. This is very variable with the site of the injury, but if phonation is found deficient, we shall find a paralysis of the pharynx, larynx, and fauces. The palate will be paralyzed, and the speech is nasal, it will be difficult for the patient to swallow, and respiration will be difficult. The pharynx and larynx will usually be anesthetic. If the lesion is paralytic the heart will be accelerated, but if irritative only, it will be retarded. The ineffective respiration will predispose the patient to aspiration-pneumonia, or hypostatic congestion. There may be increased appetite with pain in the stomach, or an absence of hunger. There are thirst and vomiting. Diabetes may supervene. When spasmodic cough, embarrassed respiration and an alteration of voice are present, with or without other symptoms, we should be apprehensive of vagus paralysis. Sometimes in diseases located in the medulla, and at times in hysteria, we find complete aphonia, stridor on inspiration, and dyspnea, and then we know that the paralysis is bilateral. It has been seen in neuroses following great shock. When it is a neurosis, phonation is more affected than respiration, since respiration is more removed from cerebral control than is phonation. In the neuroses, tachycardia, asthma, and partial aphonia have all been observed.

TREATMENT. This must be largely causal. We should antidote syphilis, remove intoxicants, stop alcohol and tobacco, and give a nutritious diet, and good hygienic directions. Massage of the larynx is beneficial in cases showing local symptoms of the parts, and electricity may be of use to stimulate the centers. Tracheotomy may be necessary.

ELEVENTH (SPINAL ACCESSORY) NERVE. As has been said, the accessory part is combined with the vagus, or 10th. The spinal part is the motor nerve to the sterno-cleido-mastoid and the trapezius, of which it especially controls only the upper portion. The accessory portion arises in the same line with the roots of the pneumogastric, and forms four or five small fasciculi which run upward, and unite with the pneumogastric in the jugular foramen. These fibres go to the pharynx, and larynx, and form an essential part of the recurrent laryngeal nerve, and so are the prominent source of the motor part of the vagus. The spinal portion arises from a long nucleus which extends down the cord as low as the sixth or seventh cervical roots in the lateral column of the cord. They issue from the cord between the ligamentum-denticulatum and the posterior nerve-roots in a number of fasciculi. These form a cord which enters the cranium with the accessory portion. Beneath the jugular foramen it leaves the accessory part, bends backward, usually superficially, to the internal jugular vein, and enters the sterno-cleido-mastoid muscle which it supplies, and then emerges and enters the trapezius just above the clavicle. It is joined here by fibers from the third and fourth cervical, forming the subtrapezial plexus, supplying that muscle. It is affected by any of the usual causes of nerve-inflammation which occur in the neighborhood of the roots of the cervical spinal nerves. It is subject to primary inflammations, which may be either unilateral or bilateral. A total paralysis of the nerve may only partially paralyze the muscles.

SYMPTOMATOLOGY. The paralysis of the two muscles to which it is distributed is the most common symptom. Little deformity results, but if unilateral, the head cannot be turned to the affected side in a backward direction. It is a form of torticollis, and if both sides are affected the head will fall backward.

TREATMENT is general.

TWELFTH (HYPOGLOSSAL) NERVE. This arises from a long nucleus in a line with those of the 3d, 4th and 6th, extending the whole length of the medulla. The cortical center adjoins that of the lips, in the lower portion of the ascending frontal gyrus. The nerve emerges from the medulla in ten or twelve fasciculi in a groove between the olive and the anterior pyramid. It passes out through the dura in two bundles by way of the anterior condyloid foramen, curves forward under the occipital artery, and reaches up to the under side of the tongue. It is liable to injury from vertebral growths, caries and injuries to the spine, from vascular dilatations and growths in the soft tissues, and diseases in the medulla. On account of the fact that its cortical fibres run to the medulla in close proximity to those to the extremities, it is extremely liable to accident in the case of a bulbar apoplexy. Disease in the posterior cranial fossa implicates it, and aneurisms of the vertebral artery.

SYMPTOMATOLOGY. Paralyses of the tongue. A hemi-paralysis is a common incident of hemiplegia; if due to an injury or disease in the medulla it is usually accompanied by the paralysis of other cranial nerves. A hemiatrophy of the tongue probably never comes from a toxic cause, but it is a common incident in tabes, syringomyelia, multiple sclerosis and the atrophy of other cranial nerves, and it is found in facial hemi-atrophy. Peripheral paralysis of this nerve produces paralysis and atrophy of one side of the tongue, the deviation is toward the paralyzed side, there is incomplete reaction of degeneration, and speech may be only slightly disturbed. It may be slight in complete hemiplegia of the tongue. Recovery is quite possible, since the paralysis is often due to a removable cause.

Electrical Treatment. The method of treatment of diseases of these nerves will depend not only on the cause, but also on the location of the lesion. If it is in the brain, the positive pole must be placed over the motor areas on the cortex for these nerves, while the other pole is to be placed upon the motor points of the muscles. If the lesion has been due to an effusion of blood, or serum, absorption will take place more readily under the influence of the positive pole. If the lesion has been a destructive one, we cannot hope for the regeneration of cells from any form of electrical treatment, but owing to the fact that every muscle is innervated by a very large number of cells, it may be possible that enough of them have escaped damage to give some stimulus to the muscle, and electricity will develope the nutrition of these cells to its fullest extent. This same rule for polarity should apply to localization of injuries in the nuclei of the nerves in the medulla, or pons. In these cases the positive should be applied to the neck at a point as near the site of origin of the nerves as is possible, while the negative is applied over the motor points of the muscles, or areas of sensory innervation on the skin. Electrical treatment of the motor oculi is of doubtful efficacy, but has some testimony in its favor. The direct method is to apply a mild negative current to each muscle by pressing a knob electrode into the orbit at the point of insertion of the given muscles, the anode at the back of the neck. Another method is to place the cathode over the closed eye, and the anode, as before, on the back of the neck. Still another method is to place both electrodes over the temples, or in the vicinity of the paralyzed muscles. Except when treating the muscles by electrode in the orbit, pass a current for two or three minutes, and strong enough to contract the facial muscles. In the degenerative conditions of the sclera, which are the result of injuries or diseases of the 5th nerve, the local application of galvanism has a stimulating effect, and it seems to aid in recovery to a very great degree. If in a lesion of any of these nerves, after the injury or disease, the reaction of degeneration is present for more than a month or two, the prospect of recovery of the nerve is very slight. On the other hand, if the nerve speedily responds to faradism, we can feel assured that its recovery is only a matter of time, and when such is the case the faradic will often give fully as good results as the galvanic current.

ANATOMY OF UPPER PLEXUS.

In considering this subject we must rely for a diagnosis of the site of injury upon two factors, viz., the exact location of the motor palsy and of the sensory losses. Some divisions of a spinal nerve are motor only, while others are as exclusively sensory, but for the most part, they are mixed in function. If we, however, find an area insensitive which is subserved by a nerve which is exclusively sensory, and at the same time discover a motor palsy in an area whose nervous supply is exclusively motor, we are at once made sure that the lesion is at some point common to both of these nerves. Carried to its full signification this divides our investigations into the two natural divisions of palsies of separate nerves, and of the brachial plexus, as a whole, or in part. In order to make the subject clear in the least possible space it is advisable to first describe plexus paralyses, since, by that method, the anatomy and pathology of the greater will also comprise that of the less.

STRUCTURE OF THE BRACHIAL PLEXUS. The anterior branches of the fifth to the eighth cervical nerves unite with the anterior branch of the first dorsal to form the brachial plexus. The roots are increased in size as they go downward, and are placed between the points of origin of the scalenus medius and anticus. The scalenus anticus arises from the anterior tubercles of the transverse processes of the third, fourth, fifth and sixth cervical vertebræ, and is inserted into a tubercle on the upper surface of the first rib. The scalenus medius arises from the posterior tubercles of all the cervical vertebræ, and is inserted into the upper surface of the first rib, just posterior to the insertion of the scalenus anticus. The three upper roots are descending in direction, the eighth cervical is horizontal, and the first dorsal is ascending, and they all unite at an acute angle. The plexus lies between the anterior and the middle scaleni, the three upper roots being above, and the two lower being behind the subclavian artery. They are thus exposed to damage by dilatations of this artery. Remember that the seventh cervical segment of the cord lies at the level of the top of the shoulder when the arm is extended. The plexus now descends obliquely through the supra-clavicular fossa, passes under the clavicle and the subclavian muscle, and is covered by the pectoralis minor and major. In this position it reaches the axilla, where it lies between the subscapularis and the serratus anticus. The axillary artery lies below the clavicle, just in front of the median portion of the plexus, and in the axilla it passes through the two divisions of the median

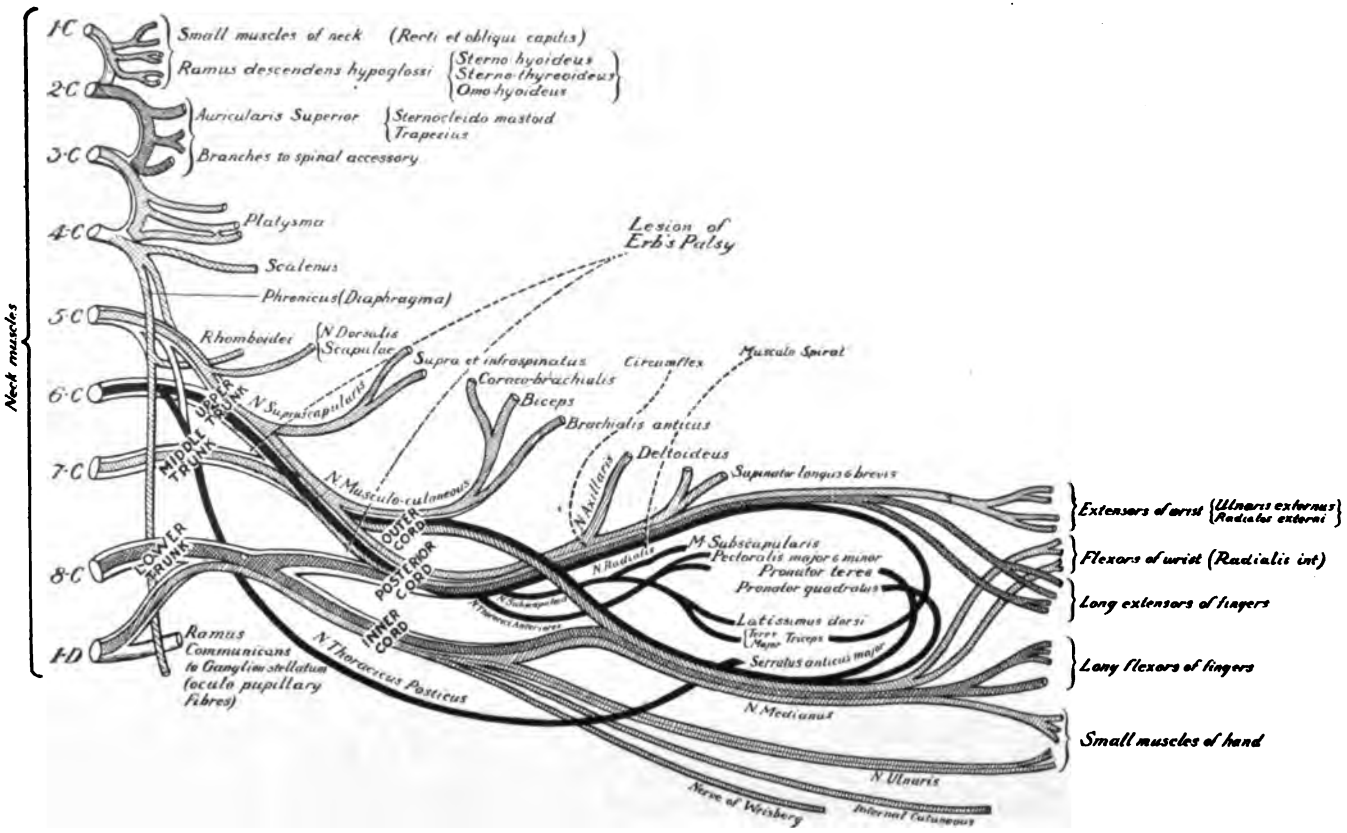


FIGURE 55. THE BRACHIAL PLEXUS. (Modified from Kocher.)

nerve, and gains a position to the rear of the plexus. Variations in this position are sometimes found to exist.

The details in the formation of the plexus are these: The fifth cervical and the sixth unite to form the upper trunk. The seventh cervical alone forms the middle trunk, while the eighth cervical and the first dorsal form the lower trunk, all these unions occurring on the surface of the scalenus medius, and covered by the scalenus anticus. Descending now, and just before reaching the clavicle, these primary trunks each throw of an anterior and posterior branch which combine in the following order to form three so-called cords, named according to their relation to the axillary artery. The anterior branch of the upper trunk, and the like branch of the middle trunk, form the outer cord; the posterior branch of the upper trunk, and the like branch of the middle and of the lower trunks, make up the posterior cord; while the anterior branch of the lower trunk alone furnishes the fibres to the lower cord. At the level of the axilla these cords break up into their final divisions.

The primary branches from the brachial plexus are divided into the supra- and infra-clavicular.

SUPRA-CLAVICULAR. Nerve to rhomboids (5 C). Arises from 5 C nerve before its union with others; nerve to subclavius (5 C). Suprascapular (5 C) sends a twig to the shoulder joint, and then divides and supplies the supra- and infra-spinatus. Posterior thoracic (5, 6, 7 C), (external respiratory nerve of Bell). This is made up by the union of the roots from 5, 6, 7 C. The upper two roots give off twigs to the serratus magnus (upper part), and the parts uniting supply the serratus magnus; the three lower cervical supply the scaleni and the longus colli.

INFRA-CLAVICULAR. External anterior thoracic (6, 7 C) goes to the pectoralis major. Internal anterior thoracic (8 C, 1 D) goes to the pectoralis major and minor. Upper subscapular (5, 6 C) goes to the subscapularis. Middle or long subscapular (7 C), goes to the latissimus dorsi. Lower subscapular (5, 6 C) goes to the subscapularis, and principally innervates the teres major.

The outer cord divides into the musculo-cutaneous and the outer root of the median. The musculo-cutaneous (5, 6 C) goes to the coraco-brachialis, biceps, brachialis anticus. After giving off these branches it divides into the anterior cutaneous and posterior cutaneous, both going to the outside of the forearm.

The posterior cord divides into the circumflex and musculo-spiral.

The circumflex (5, 6 C) sends a twig to the shoulder joint, and then divides into two branches. Superior: the upper division is cutaneous; the lower goes to the deltoid. Inferior: the upper is cutaneous; the lower goes to the teres minor.

Musculo-spiral (6, 7, 8 C) branches. Internal cutaneous to the inside of the arm; upper external cutaneous goes to the outer side of the arm; the long external cutaneous to the outer side of the arm, and back of the forearm. It now divides into the radial and posterior interosseous. The radial is the cutaneous branch, and goes to the dorsal surface of the thumb, and the first and radial half of the second finger. Posterior, interosseous branch goes to the extensor carpi radialis brevior, extensor ossis metacarpi pollicis, extensor indicis, extensor communis digitorum, extensor minimi digiti.

The median nerve (6, 7, 8 C, 1 D) goes to pronator radi teres, flexor carpi radialis, flexor sublimis digitorum, flexor profundis digitorum. It then sends out a branch called the anterior interosseous, which supplies the pronatus quadratus; and another branch to the flexor longus pollicis; then is sent off the palmar cutaneous which goes to the palmar surface of the thumb, 1st, 2d and radial half of the 3d finger, palm almost to the wrist. It is the cutaneous supply, not only to the palmar surface of the thumb, of 1st, 2d and radial half of 3d finger, but also to the dorsal aspect of the last phalanx of all these.

The muscular branches go to the opponens pollicis, abductor pollicis, flexor brevis pollicis, outer half, 1st lumbricalis to 1st finger; 2d lumbricalis to 2d finger; outer half of 3d finger.

Inner cord. This first throws off the internal anterior thoracic to the pectoralis major and minor; nerve of Wrisberg; a cutaneous branch to the olecranon and the lower third of arm—small internal cutaneous (8 C, 1 D). It is divided into the anterior (or external posterior), and internal. The main stem then proceeds as the ulnar.

The ulnar (8 C) throws off a twig to the elbow joint; a branch to the inner part of the flexor profundis digitorum; a branch to the flexor carpi ulnaris; two inches above the wrist joint it gives off the dorsal cutaneous to the dorsal surface of little, 3d, and ulnar half of 2d fingers; at the middle of forearm gives off the palmar cutaneous to the ulnar half of palm.

The terminal branch of the ulnar supplies the inner surface of the little, and ulnar half of 3d finger; the deep branch supplies inner half of the flexor brevis pollicis, the abductor brevis pollicis, and then terminates in a group of branches which supply the abductor minimi digiti, the opponens minimi digiti, the flexor brevis minimi digiti, 4th dorsal interosseous, 3d palmar interosseous, 4th lumbricalis, 3d dorsal interosseous, 2d palmar interosseous, 3d lumbricalis, 2d dorsal interosseous, 1st palmar interosseous, 1st dorsal interosseous.

NERVE SUPPLY TO MUSCLES OF THE ARM AND HAND.

Muscles Which Move the Arm. Abductors move the arm away from the body: Deltoid; Supraspinatus.

Deltoid (circumflex) moves the arm upward, outward, forward, or backward, according as the middle, anterior, or posterior division of the muscle is contracted. When it is paralyzed the shoulder can be raised, but the arm cannot be abducted, nor elevated backward or forward. The shoulder is flat, and there is a groove between the acromion and the head of the humerus as it is subluxated. Each division of the deltoid may be paralyzed separately.

Supraspinatus assists in the elevation of the arm, and when paralyzed, the space between the acromion and the humerus is greater than when the deltoid alone is paralyzed.

Adductors which bring the arm in to the thorax: Pectoralis major (int. and ext. ant. thoracic); Coraco-brachialis (musculo-cutaneous); these are also flexors. Latissimus dorsi (long subscapular), Teres major (lower subscapular); these are also extensors.

Pectoralis major draws the arm to the thorax, the clavicular portion helps to depress the elevated arm to a horizontal position, and then inward. With the arm at rest, it draws the acromion upward and forward. The sternal portion draws the arm from the vertical position downward, and when the arm is at the side moves the acromion forward and downward. If it is paralyzed adduction of the arm is imperfect, and paralysis is best discovered by extending the arms, and attempting to press the volar surfaces against one another.

Coraco-brachialis adducts and flexes the arm, and hence these two functions will be impaired if it is paralyzed.

Latissimus dorsi draws the elevated arm backward and downward. When the arm is at rest it draws it inward and backward, but acting alone it pulls the dorsal end of the trunk to one side, but if it acts conjointly it extends it. If it is paralyzed the arm cannot be moved upward, the trunk cannot be moved laterally, and the dorsal spine is insufficiently extended.

Teres major adducts, extends, and rotates the wrist and the humerus inward. It adducts the arm to the thorax if the shoulder is fixed, and slightly elevates it. Paralysis is not conspicuous, as its function is assumed by other muscles.

Outward Rotators. Infraspinatus (supra-scapular), Teres minor (circumflex), Subscapularis (upper and lower subscapular).

Infraspinatus and Teres minor rotate the arm outward, and if paralyzed the arm cannot be rotated outward, and there is difficulty in writing.

Muscles Which Move the Whole Forearm. Flexors: Biceps (musculo-cutaneous), Brachialis anticus (musculo-cutaneous), (musculo-spiral slightly), Brachio-radialis (musculo-spiral).

Biceps flexes the forearm; if the hand is pronated the biceps supinates it. The second tendon of insertion tightens the fascia of the forearm. If it is paralyzed flexion is imperfect, but the movement is carried out partially by other muscles.

Brachialis anticus also is a flexor of the forearm, and its paralysis will partially embarrass flexion.

Brachio-radialis acts mostly as a flexor of the forearm, but when the hand is prone this muscle is capable of nearly half-supinating it; after strong supination it will partially effect pronation. Paralysis will therefore be slightly evident in loss of function.

Extensors. Triceps and Anconeus (both musculo-spiral).

Triceps extends the forearm; the long head of the triceps, and the coraco-brachialis help to keep the head of the humerus in position. If paralyzed the arm cannot be extended, except by its own weight. If the long head of the triceps is paralyzed subluxation of the head of the humerus readily occurs.

Anconeus extends the forearm.

MUSCLES MOVING THE OUTER PART OF THE FOREARM. Pronators. Pronator radii teres (median), Pronator quadratus (anterior interosseus branch of the median).

Pronator radii teres pronates the hand by moving the radius, to which the hand is articulated; also slight flexion of the forearm.

Pronator quadratus pronates the radius, and therefore the hand. Paralysis of these two muscles will therefore paralyze pronation, and paralysis of one of them affects it partially.

Supinators. Supinator longus and brevis (musculo-spiral).

Supinator longus flexes the forearm, and aids in supination. When it is paralyzed flexion and supination are imperfect, the muscle does not stand out prominently if the arm is flexed, or on attempt by another to extend it forcibly. If the muscle is atrophied the forearm is spindle-shaped.

Supinator brevis supinates the hand when the forearm is extended, and if paralyzed there is imperfect supination of the hand.

Flexor carpi radialis (median), Flexor palmaris longus (median), Flexor carpi ulnaris (ulnar). These are all flexors.

Flexor carpi radialis flexes and slightly pronates the hand; in conjunction with the Extensor carpi radialis longus it abducts the hand. If paralyzed there is imperfect flexion.

Palmaris longus tightens the fascia of the palm, and then flexes the hand. The flexion is thus of the wrist only, and if paralysis occurs flexion is impaired, but there is no unusual position of the hand, because it falls by its own weight. The flexors of the fingers may act as a substitute.

Flexor carpi ulnaris flexes the hand, supinates it, and in conjunction with the Extensor carpi ulnaris it adducts the hand.

Extensors. Extensor carpi radialis longus, Extensor carpi radialis brevis (both musculo-spiral), Extensor carpi ulnaris (post. interosseus branch of musculo-spiral).

Extensor carpi radialis longus extends the hand, and in conjunction with the Flexor carpi radialis it abducts the hand. The brevis has the upper extension function only. If paralyzed the wrist cannot be flexed dorsally, i. e., extended or abducted, and there is flattening of the forearm.

Extensor carpi ulnaris extends the hand, and in conjunction with the Flexor carpi ulnaris it adducts the hand. If paralyzed the wrist cannot be flexed dorsally, or adducted. There is wrist-drop, which is characteristic of paralysis of the extensors. The palmar pressure is weak because the flexors of the fingers can only be brought into complete action when the hand is extended.

MUSCLES MOVING THE FINGERS AND THE FIFTH METACARPAL BONE. Flexors. Flexor sublimis digitorum (median), Flexor profundus digitorum (ulnar), Flexor ossis metacarpi minimi digiti, Flexor brevis minimi digiti (both deep branch of the ulnar), Lumbricales (outer two, median; inner two, ulnar).

Flexor sublimis digitorum primarily flexes the second phalanges; secondarily flexes the first phalanges. If paralyzed the second phalanx can from overweight of the extensors gradually be flexed toward the first, and even subluxated.

Flexor profundus digitorum flexes the third phalanx primarily, after this flexion of the second, and slightly of the first. The second and third phalanges cannot be flexed in case of its paralysis, and there may be subluxation between the second and third phalanges, but it is very rare as an isolated paralysis.

Flexor ossis metacarpi minimi digiti flexes the fifth metacarpal bone, and slightly adducts it toward the middle of the hand.

Flexor brevis minimi digiti flexes the first phalanx of the middle finger.

Lumbricales flex the first phalanges, and extend the second and third. The first and second slightly adduct the index and middle fingers from the middle line, the third and fourth slightly adduct the ring and little fingers to that line. For result of paralysis see Interossei.

Extensors. Extensor communis digitorum, Extensor minimi digiti (both musculo-spiral), Extensor indicis (musculo-spiral, post. interosseus branch).

Extensor communis digitorum extend the fingers, the oblique branches at the sides of the ring finger, greatly limiting the extension of the ring finger when its neighbors are flexed. With the Extensor minimi digiti and the Extensor indices they extend the first phalanges of all the fingers, and abduct them. If they are paralyzed the first phalanges cannot be extended, nor can the fingers be abducted. The grasp is weak, because the flexor muscles are shortened, and cannot contract forcibly.

Abductors. Interossei dorsales (deep branch, ulnar), Abductor minimi digiti (ulnar).

Interossii dorsales each abduct from a line coinciding with the long axis of the middle finger. This digit, having two interossei inserted into it, is abducted toward the radial side by one, and toward the ulnar side by the other; thus each in turn becomes an adductor, for the finger being abducted by one, is adducted by the other. They also flex the first phalanges, and extend the second and third.

Abductor minimi digit abducts the little finger.

Adductors. Interossii palmaris (deep branch of ulnar).

Interossii palmaris each adducts its digit toward the middle finger; also it flexes the first phalanx of its digit, and extends the second and third phalanges.

The interossii and lumbricales adduct and abduct the fingers, if the first phalanges are extended, with flexion of the first phalanges and simultaneous extension of the second and third phalanges. If paralyzed the fingers cannot be adducted, nor abducted. The interosseus spaces are very marked, there is "main en griffe," due to extension of the first phalanges, and flexion of the second and third phalanges.

Muscles Moving the Thumb and Its Metacarpal Bone. Flexors. Flexor ossis metacarpi pollicis (median), flexor brevis metacarpi pollicis (median), flexor longus pollicis (ant. interosseus branch of median).

Flexor ossis metacarpi pollicis flexes and inwardly rotates the first metacarpal bone.

Flexor brevis metacarpi pollicis flexes the first phalanx of the thumb.

Flexor longus pollicis flexes the second phalanx of the thumb. Paralysis prevents this motion, and hinders writing. These muscles of the ball of the thumb, which are inserted in the radial side of the first phalanx and metacarpal bone, have the function of moving the first metacarpal forward and backward, and of flexing the first phalanx, and rotating it, so as to appose the thumb to the fingers.

Extensors. Extensor ossis metacarpi pollicis (post. interosseus branch musculo-spiral).

Extensor brevis pollicis, extensor longus pollicis (all innervated by post. interosseus branch of the musculo-spiral).

Extensor ossis metacarpi pollicis extends and abducts the metacarpal bone of the thumb.

Extensor brevis metacarpi pollicis extends the thumb. It is the abductor of the thumb. It brings the first metacarpal directly outward, extends the first phalanx, but has no influence over the second. Paralysis of the muscle is of importance only when the Abductor longus pollicis is paralyzed at the same time.

Extensor longus pollicis extends both phalanges of the thumb. This latter also moves the metacarpal bone outward, and at the same time forward, i. e., flexes it toward the wrist-bone, and at the point of greatest contraction is a flexor of the hand. If the abductor longus pollicis and the extensor brevis are paralyzed, the thumb is adducted, and a volar hand is produced.

Abductor. Abductor pollicis (median).

Abductor pollicis abducts the thumb.

Adductor. Adductor pollicis (ulnar).

Adductor pollicis adducts and flexes the thumb.

UPPER PLEXUS INJURIES.

Total plexus-paralysis is a possibility, and it may be from disease of the cells of the nuclei of origin, or of the roots, or of the plexus in continuity. It is often impossible to exactly fix the site of damage and rarely is it of importance so to do. Accidents affecting the whole plexus are not uncommon, and are the usual cause of total plexus-paralysis, since a neuritis is very rare with such a localization. Fracture of the humerus or clavicle, or the resulting callus, subcoracoid, or subglenoid, dislocation of the humerus, or hemorrhage, may all produce this form of injury. Such accidents are common in delivery, and sometimes occur in attempting to reduce dislocations. They may be from tumors in the upper clavicular region, or aneurisms of the subclavian. In these accidents, the ulnar most often escapes. After emerging from the plexus, the nerves are always affected singly.

SYMPTOMATOLOGY. After being complete for a short time, it is apt to recede from all but a few nerves. The motor palsy is complete at first, but the sensory is generally incomplete, so soon as the primary shock disappears, and the inner part of the upper arm is apt to escape entirely on account of the vicarious innervation which it receives from the intercosto-humeral nerve. The paralysis is always degenerative.

PROGNOSIS. This is grave, but improvement should not be despaired of for a very long time, as it may slowly improve for many years.

TREATMENT. This is mainly surgical, as the plexus is either ruptured by the violence of the causative injury, compressed by a new-growth, or vascular dilatation, or imprisoned in connective-tissue or callus

ERB'S PARALYSIS (DUCHENNE'S). This is the most important of the partial paralyses of the plexus, and is of the upper-arm type, as distinguished from the lower-arm type called Klumpke's. This affects the shoulder, and arm in its upper part, on account of an injury to the roots of the 5th and 6th cervical. This may result from forcibly pressing the clavicle against the vertebral column, or the first rib may be the instrument causing the pressure. This condition may be the result of holding the arms upward, outward and backward for a considerable time in surgical positions, and is more liable to produce the injury if the head is, at the same time, turned to the opposite side. The narcosis from anesthetics is also a contributing cause, since the whole organism is, to a degree, in a condition of intoxication similar to that from alcohol, which we know is an essential element in the production of "Saturday Night Palsies," seen in those intoxicated persons who have fallen asleep with the arm hanging down, or flexed under the head. It is common in infants with a history of traumatism during delivery, and there is, with the palsy, a local anesthesia in the distribution of the circumflex nerve. It may be produced by occupations where heavy burdens are carried on the shoulder, and is here more liable to occur if the person is also affected by some one of the cachexiæ, as tuberculosis. It is almost always from injury, but has been the result of primary intoxications; rheumatism, infections, and ptomaine poisoning have caused it. From intoxications it is bilateral, while from injury it is unilateral. It always involves the deltoid (the nerve is the circumflex), biceps (musculo-cutaneous), brachialis internus anticus (musculo-cutaneous), supinator longus (musculo-spiral), often the supinator brevis (mucsulo-spiral, posterior interroseous branch), occasionally the supra- and infra-spinatus (suprascapular), and subscapularis (upper and lower subscapular), and the clavicular end of the pectoralis major.

The deltoid abducts and raises the arm, but alone draws the arm forward, while the scapular portion draws the arm back. The biceps flexes the forearm, as does the brachialis internus, while the coraco-brachialis adducts and flexes the arm. Supinator longus turns the forearm to a supine position, while the supinator brevis, which is wrapped about the head of the radius, also supinates the hand. The infraspinatus rotates the arm outward, and the subscapularis rotates it inward. If the supinator brevis is involved, the whole lower arm and hand are in a state of pronation, and cannot be satisfactorily supinated.

SYMPTOMATOLOGY. The paralysis is almost always atrophic, and the reaction of degeneration is present to a variable degree, and the shoulder hangs lower, so that there may be a grade of subglenoid dislocation. When the partially paralyzed muscles are put into action, we may find fibrillation. Pain may be present, but it is never pronounced; in many cases sensibility is intact, even in the region of the axillary nerves, or it may be very transiently abolished. In severe cases, its distribution is as follows: there is anesthesia in the distribution of the circumflex, and musculo-cutaneous nerves, the outer surface of the upper-arm above the median portion of the deltoid, not quite up to the acromion, and on the outer side of the forearm; there is sensory loss at times in the fibres of the median, going to the thumb and the index finger.

KLUMPKE'S PARALYSIS. A lower-arm type. This arises from an injury of the 8th cervical and 1st dorsal roots. It may possibly be produced by violence in the reduction of a dislocation, but more often arises from some disease of the first rib, or meningitis from syphilis, or operative injuries. Primary neuritis is sometimes a cause, or it may be the final residuum of a total plexus injury. It is a rare form.

It is a disease of the ulnar and median nerves, and as a result, while the arm can be elevated, and the forearm flexed and supinated, on account of the paralysis extension of the forearm is impossible, and the hand is totally paralyzed. Paralysis of the interossii produces an inability to spread out, or to approximate the fingers, or extend the inter-phalangeal joints. Adduction of the thumb becomes impossible, and the paralysis of the flexors of the fingers and the wrist renders the patient incapable of bending the joints, or of grasping anything in the fingers. The paralysis is atrophic, and the thenar and hypo-thenar eminences are wasted, but there are no vaso-motor symptoms. The sympathetic nerve may be involved, or spared, according as the injury is inside, or beyond the point, where the rami communicantes are given off to the cervical sympathetic from the nerve If the sympathetic is involved, there is a narrowing of the palpebral fissure, contraction of the pupil, sinking in of the eyeball, and wasting of the face on that side. No dilatation of the pupil results from shading it from the light, or from the instillation of cocaine, and the cilio-spinal reflex (dilatation of the pupil from pinching the skin of the neck) is absent. There are sensory losses in the region of the median, the inner surface of the forearm and upper-arm, and also in the ulnar region of the hand. These may be incomplete.

TREATMENT. Rest of the part, massage, and electricity are the essentials. Rest should be so devised as to avoid strain to the nerves, and when the injury is about the shoulder joint, the arm should be supported in a sling. The extensors of the wrist and fingers should be supported by a splint. Massage of the muscles is always useful. If the muscles will respond to faradism, it may be used, but galvanism is of greater benefit, since it stimulates nutrition more efficiently. Use a current strong enough to produce contractions, 15 to 20 to each muscle, over the motor point, using that pole which gives the best contractions. It is a good plan to give one or two treatments daily. For pain use heat in any form. Keep the part always well wrapped up. Rest in nerve injuries is more important than in paralyses. A mild, constant galvanic current often relieves pain. Use counter-irritation if the pain persists. Mustard and iodine are good, but the actual cautery is the best. Analgesics of the coal-tar series may be needed, while change of scene and climate are often of surprising benefit. Suture is the only remedy for divided nerves. As soon as the diagnosis can be made, it is best to operate. In continued cases it may be necessary to free the nerve from the connective tissue which has engaged it. If end to end approximation cannot be done, attempt to connect by the implantation of animal nerves, or by tubes of decalcified bone. Injuries to sympathetic nerves can rarely be benefitted by treatment. In obstetrical palsy of Erb's type, improvement in position, and some gain in function have followed late operation.

INDIVIDUAL BRACHIAL PALSIES.

Paralyses of individual nerves may be due to disease of the nerve-roots, of the plexus, or of one of the nerve-trunks from the plexus. The only important nerve arising from the roots themselves is the posterior thoracic. The musculo-spiral and the median are most usually the ones affected by the pressure palsies, with the added influence of some chronic intoxicant. Secondary forms occur at times, chiefly when the primary neuritis has been from an infection, and the ulnar nerve is the one most commonly primarily affected, out-numbering the other two. Traumatism is the most common cause, but it may also follow toxic and infectious influences, gout, rheumatism, grippe, and the intoxications of gestation. Tumors, aneurisms, exostoses may all excite it from pressure. Most common in women, and in persons over forty years of age. While paralysis of the whole plexus is rare, a neuritis of single nerves is common. The reaction of degeneration is present in the muscles, from the fact that the integrity of the nerve-endings in them is compromised whenever the nutrition of the nerves is impaired.

Long Thoracic (5, 6, 7 C.) is the nerve to the serratus, and is also called the respiratory nerve of Bell. Its paralysis most often results from injury, gunshot wounds, or excessive strain from exertion with the arms in an uplifted position. It has occurred after the puerperium from muscle-strain during labor. Some cases seem purely toxic, as in infections like typhoid, grippe, diphtheria; it has been hysterical (possibly). On account of its etiology it usually occurs in muscle-workers, males of the age of twenty-five to forty, and the right side more often than the left. It is not usually involved in upper plexus-paralysis.

Symptomatology. These are of serratus paralysis. The function of this muscle is to hold the scapula against the thorax, and to rotate it in raising the arm. This paralysis weakens all movements which require fixed positions of the scapula. Inspiratory movements on that side are reduced, but there is no actual paralysis. If from a neuritis there is pain in the neck and shoulder, and sensation is usually disordered. In severe cases there is the reaction of degeneration. The motor points are on the sides of the chest below the axilla.

Diagnosis. This rests upon the fact that at rest the lower angle of the scapula is nearer the spinal column than is normal, and if the arm is raised, the lower angle of the scapula swings out, and is displaced upward by the unopposed action of the levator anguli scapulæ and the rhomboids ("angel-wing scapula").

Prognosis. It is recovered from in only about one-third of the cases. In any case recuperation is tedious, and may take months in a slight case.

Treatment. The arm should be supported, and the strain should be taken off the scalenius, which means that no movement should be allowed which involves raising the arm. Since the chance of recovery is so doubtful, it has been recommended that the pectoralis major should be split, and a portion of it inserted into the serratus. If local treatment is to be applied it should be over the scalenius, since the nerve is most accessible at this point.

Suprascapular (5 C.). This is distributed to the supra- and infraspinatus. Traumatic generally, and may be produced by falls upon the shoulder, dislocation of the humerus, and the pressure of heavy burdens. The principal loss is the power of outward rotation of the arm, and some movements requiring great dexterity in the manipulation of the arm as a whole. The paralysis of the supraspinatus simply impairs the action of the deltoid to some extent. It does have some power in rotating the shoulder inward. The only sensory change is a small spot of anesthesia over the scapula. Circumflex usually involved.

Symptomatology. Rotation of the arm outward is impaired, and somewhat on inward rotation, but the office of these muscles may, to some extent, be assumed by the deltoid and the teres minor. Both these muscles may show a little hypertrophy.

Diagnosis. This rests upon faulty rotation, difficulty in writing sometimes, anesthesia in a small spot over the scapula, and hypertrophy of deltoid and teres minor.

Treatment. As above; postural and electrical.

Circumflex (5, 6 C.). This supplies the deltoid, teres minor, and goes to the third head of the triceps, and the skin over the lower part of the deltoid. It is usually from falls on the shoulder, or on the hand, from crutch pressure, from continued elevation of the arms in sleep, from rheumatism and various intoxications, and cachexias combined with pressure.

SYMPTOMATOLOGY. The paralysis of the deltoid may not be complete, since the muscle receives some of its supply from one of the thoracic nerves. The deltoid atrophies so that the acromion is exposed. Since the nutrition of the shoulder-joint comes through this nerve, we are liable to find adhesions, and some degree of anchylosis. The sensory symptoms may be slight, but typically there is anesthesia of the skin over the deltoid, and there may be hyperesthesia in other parts of its distribution, and there is pain and tenderness along the course of the nerve. The reaction of degeneration occurs quite early, excepting in cases arising from contusion. A sub-glenoid dislocation is usually present.

DIAGNOSIS. This is usually easy from the flattened appearance of the shoulder with the prominence of the point of the acromion, except in a fat person. Here the partial dislocation of the humerus is a guide. The arm cannot be abducted. When anchylosis is a symptom, there must be a differentiation between it and a rheumatic or traumatic anchylosis, where a neuritis has been produced in addition. In these two latter contingencies the scapula will move when the arm is passively moved, and it will not do so in case of a paralysis. In case of a muscular paralysis from blows upon the muscle there will be no anesthesia, nor reaction of degeneration.

TREATMENT. As above.

MUSCULO-CUTANEOUS (5, 6 C.). Trauma, pressure, and dislocation of the head of the humerus are the chief causes of this palsy. The musculo-cutaneous supplies the chief flexors of the elbow, and the skin on the radial side of the forearm, the distribution of the lateral cutaneous. It is rarely paralyzed alone. The brachialis is often spared, since it is supplied by the branches of the musculo-spiral, and the coraco-brachialis is also spared at times, so that a paralysis of the biceps alone has been observed.

SYMPTOMATOLOGY. In general the arm cannot be flexed in a supinated position. It is only possible in a prone position, where the supinator longus is the efficient muscle. There is rapid atrophy on the outer surface of the upper-arm between the insertion of the deltoid and the origin of the supinator longus. There may be some grade of anesthesia on the radial side of the front and the back of the forearm. Reaction of degeneration is very early present.

PROGNOSIS. Recovery is the rule, and in a moderate case has resulted in three months.

MUSCULO-SPIRAL (6, 7, 8 C.). Supplies the triceps, all the muscles on the back of the forearm, the extensors of the wrist and the fingers, both the supinator longus and brevis, the skin on the radial side of the back of the hand, the thumb, index finger and the radial half of the middle-finger. Most subject to paralysis of any of the individual nerves, on account of the fact that as it winds about the outside of the back of the humerus and the external condyle it is unprotected by any great amount of muscular tissue, and rests upon hard bone. In the upper-arm it is between the triceps and the humerus, and lower down between the brachialis anticus and the supinator longus. Traumatism is almost always the cause, contraction of the triceps in forcible contraction of that muscle has caused it, as in pulling on a boot, crutch pressure, and strains and blows. Infection and intoxication have been doubted, except as accessory to trauma. If the system has been chronically poisoned by any intoxicant or infection, pressure may localize its effects in this exposed nerve, and this is the usual conclusion in regard to "Saturday-night" palsies, or those from narcosis, or the hypodermic administration of various drugs.

SYMPTOMATOLOGY. In crutch palsies the lesion involves the branch to the triceps, but ordinary pressure cases affect the nerve below this point, and the triceps escapes, and the supinators also. Commonly we find a paralysis of the extensors of the elbow and the wrist, the long extensors of the fingers and thumb, and often of the supinators. There is wrist-drop (spastic), and commonly the paralysis is greatest in the index, and decreases regularly in grade to the little finger (inexplicable). The arm cannot be supinated, and if an object is firmly grasped the contraction twists the arm into a position of pronation. This comes from the paralysis of the supinators, especially the longus. Power of flexion of the forearm is decreased from the paralysis of the supinators. If the paralysis lasts a long time we find Gubler's tumor at the wrist. This is said to be due to either a thickening of the sheaths of the tendons, or to a partial dislocation of one of the bones of the carpus. The loss of sensation is a variable one, since the common site of injury is near the musculo-spiral groove, when it affects sensation only so far as to produce a tingling in the hand as a rule. If the injury is above that level, anesthesia and hyperesthesia will be present in the lower half of the arm, the back of the forearm, the back of the thumb, and of the first three fingers. If it is a crutch-palsy there is anesthesia chiefly on the front of the arm, in the area of distribution of the external and internal cutaneous, but there may be hyperesthesia and anesthesia. In compression cases there is little change in the electrical reactions, but in the severer lesions it is marked. This nerve furnishes fibres to the wrist and carpal-joints, and therefore we may find adhesions and anchylosis.

PROGNOSIS. Generally good, and recovery often ensues in a few days to six weeks without treatment, but if there is complete reaction of degeneration, it cannot be expected for many months. Narcosis-palsy recovers as a rule in from one to four months. The amount of the reaction of degeneration is a useful criterion.

DIAGNOSIS. One fact should be pointed out in considering the diagnosis. This is the most definite instance of the paralysis of a single function, i. e., extension of the hand, and since functions, and not muscles, are represented in particular segments of the cord, we ought to bear in mind the possibility that the losses which we are considering as peripheral may be due to a spinal lesion. Otherwise the peculiarity of the loss makes the diagnosis evident. It is the nerve most commonly affected in multiple neuritis, and therefore the fact must be made certain that it is confined to one arm, and has been so from the first, and that it was sudden in onset. In multiple neuritis from lead also, the supinator longus is usually spared; the electrical reactions are more degenerative. Supinator longus paralysis is discovered by placing the forearm midway between pronation and supination. See if movement shows the belly of muscle. In progressive muscular atrophy the paralysis is rarely so complete; it involves both the radial and ulnar, and the electrical reaction, while reduced perhaps, is normal qualitatively for a long time.

TREATMENT. This is mainly postural, and the elbow should never be extended, in order to avoid injury from a contraction of the triceps. The use of electricity is advised after the following manner: Place the positive galvanic electrode on the gluteus, and the cathode on the motor points for the musculo-spiral. Electrodes should be twenty to thirty c.m. square. Increase the current until the patient in endeavoring to extend the hand feels some ease in the movement. Strength generally needed is six to eight m.a.

MEDIAN (6, 7, 8 C., I D.). This nerve goes to the pronators, the radial flexor of the wrist, the flexors of the fingers excepting only the ulnar half of the deep flexor, the muscles which abduct and flex the thumb, and the two radial lumbricales. It collects sensory impressions from the radial side of the palm, on the front of the thumb, of the first two fingers, half of the third finger and, in some persons, from the back of the last phalanx of the index and middle fingers. Sometimes also from the adjacent parts of the ring finger, and the last phalanx of the thumb.

Its cause is usually traumatism received in the patient's occupation. It may come from the long-continued and repeated grasping of an object between the hand and the upper-arm, as necessitated in some trades, from a violent contraction of the pronator radii teres, or from dislocation of the wrist. The fracture of the humerus rarely, of the bones of the forearm occasionally, has produced it. It is sometimes toxic.

SYMPTOMATOLOGY. The symptoms will differ in accordance with the site of the lesion. If injured above the wrist-joint pronation cannot be accomplished completely. After partial action of the flexors, the arm must be separated from the side, and the humerus rotated inward. If the ulnar is preserved we shall find some remaining power of flexion of the wrist and the terminal phalanges of the two inner fingers, and the first phalanges of all. The thumb is in persistent adduction, and cannot be apposed to the fingers, and it assumes a position on the same plane as the fingers ("ape-hand"). The wrist can be flexed only by a strong inclination to the ulnar side. If the paralysis is peripheral to the wrist-joint the small muscles only of the hand are paralyzed. Pain may be present in the hand. The sensory losses may be absent or conspicuous. There are sometimes atrophy and glossy skin. Electrical excitability and degeneration upon the severity of the lesion.

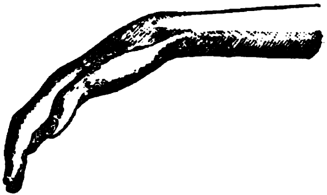


FIGURE 56. APE HAND.
(From Oppenheim.)
Courtesy of J. B. Lippincott.

PROGNOSIS. Is generally grave, as the injury is usually a serious one.

DIAGNOSIS. This depends upon the detection of a loss of the power of flexion and pronation, with the distribution of the sensory losses, if present and well-marked.

TREATMENT. As in other forms.

ULNAR (8 C., 1 D.). This nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossii, some of the lumbricales, the adductor and inner head of the short flexor of the thumb. Its sensory distribution is to the ulnar side of the back and front of the hand, but while it goes to two fingers and a half on the back, it supplies only one and a half on the front. It is superficial behind the elbow and at the wrist. It is vulnerable to the injuries and intoxications common to other nerves, and may also be paralyzed by continued extension in injurious positions while the subject has been asleep (Gordon), and has also suffered from forcible flexion of the forearm. Particularly subject to injury in emaciated persons, or those who are cachectic.

SYMPTOMATOLOGY. On account of the paralysis of the interossii it renders writing very difficult, the adduction of the thumb is lost, and so is that of the little finger, as well as other movements of that member. The fingers cannot be flexed at the first, or extended at other joints. The weakness is in the little and the ring fingers, and the loss in the others is compensated for by their lumbricales. In flexion of the wrist the hand inclines toward the radial side. The hand assumes the "claw-hand" type of deformity, which consists of over-extension of the first phalanges, and flexion of the two distal ones. This is produced by the over-action of the extensor communis digitorum and the long flexor of the fingers, because of the paralysis of the interossii and lumbricales which are their opponents. This is most pronounced in the fourth and fifth fingers. since the lumbricales of the other two are still competent from innervation by the median. Complete closure of the hand is impossible on the same grounds. Dupuytren's contraction of the palmar fascia has been a result. Sensory disturbances are variable, and may be very slight, as in cases of complete ulnar palsy the little finger, or even the ball of it, has been the sole area of sensory aberration. Since the dorsal trunk of the nerve turns to the dorsal side at the junction of the middle and lower third of the forearm, and the nerve runs between the ulna and the flexor carpi ulnaris, a cutting wound below this point, and on the palmar aspect, will not affect the sensibility of the dorsal portion of the hand and fingers. Subjective tingling may be the only symptom. Atrophy hollows out the palm and the base of the little finger, and the thumb may rotate forward slightly. This is not so marked as in progressive muscular atrophy, where all of the lumbricales are affected.

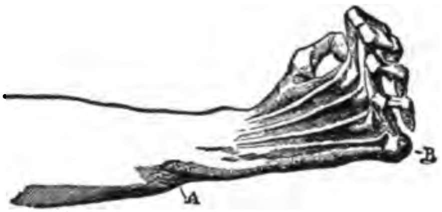


FIGURE 57. CLAW HAND. (From Oppenheim.)
Courtesy of J. B. Lippincott.

PROGNOSIS. Pressure palsies, if slight, may be recovered from in a few weeks, but severer forms may take months or years, and from injuries a recovery depends upon the possibility of repair. Infection of the plexus takes place more often in injuries of the ulnar than in the case of any other individual nerve.

DIAGNOSIS. This is evident, and the only error which might occur is in considering it peripheral, when a similar palsy is known to occur from a lesion of the cord at the lowest part of the cervical enlargement.

TREATMENT. This is the same as in other palsies of the same locality.

NEURITIS OF THE PHRENIC NERVE.

This nerve arises from the 3d, 4th and 5th C., and is not liable to any form of injury, except in the case of deep stab-wounds, or injuries from bullets. While it is not a probable cause, phrenic paralysis may arise from spondylitis, and fractures, and dislocations of the cervical vertebræ. It is also possible from tumors, and aneurisms in the neck and thorax, and from hemorrhages in the spinal canal, and the different forms of meningitis, syphilitic meningitis being a quite frequent cause. The most usual cause is a disease of the spinal cord, or its envelopes. It is occasionally involved in tabetic degenerations, and the branches distributed to the diaphragm may become affected in pleurisy and peritonitis. From traumatism and pressure usually results a unilateral form, but from intoxications, as from lead, rheumatism, as a factor in the multiple neuritis from alcohol, and after diphtheria, it is bilateral. Cold has been urged as a possible cause, and so has hysteria.

Differential Diagnosis. The diaphragm may be immovable, from injury to the Phrenic Nerve, from Disease of the Spinal Cord, from a Myositis set up by a contiguous pleuritis or peritonitis, and from hysteria.

Symptomatology. These are evidences of paralysis of the diaphragm. When the paralysis is unilateral, its detection is very difficult, yet may be discovered after repeated examination. Even when the paralysis is bilateral, there is likely to be little embarrassment of respiration, except on exertion, when respiration will become labored and the voice will become weak. On inspection it will be noticed that the thorax is in very exaggerated action, and that, on inspiration, the epigastrium will actually recede, instead of advancing. The absence of Litten's sign can often be appreciated. This consists in the fact that on inspiration a shadow will be seen to run downward from the lower border of the sixth rib, and ascend again on expiration. The observer must stand so that the bared abdomen of the patient is between the observer and a strong light. Another set of phenomena follows a loss of power in the diaphragm. There is difficulty in expectoration, defecation, sneezing, and other operations requiring abdominal effort. The sensory disturbances are obscure, and are overlooked, or misconstrued as denoting neuralgia, or muscular rheumatism.

Differential Diagnosis. In injuries to the nerve, the diaphragm suffers alone, unless the neuritis is a part of a multiple neuritis. When the nerve is affected by a neuritis, there is a painful spot on the scalene muscles, or at the lower insertions of the sterno-cleido-mastoid.

If it is from disease of the spinal cord, other muscles always suffer, and the associated condition is often overlooked, because these muscles are those which are innervated from the cervical, and not from the dorsal cord.

If it is a myositis from pleuritis or peritonitis, we shall find the symptoms of the primary disease, the action of the diaphragm will elicit the characteristic pain, and fever will be present.

If from hysteria, in which condition patients will often breathe with great rapidity for a long time, even in conditions of rest, careful watching will, after a time, detect a normal movement of the diaphragm. One such movement will settle the diagnosis. We shall often be able to find the stigmata of the disease, and, in any case, ether narcosis will clear up the condition.

PROGNOSIS. It is good in cases when evident pressure may be removed, or the results of acute injuries are amenable to treatment. It is also good in rheumatic and post-diphtheritic cases, but less so in the alcoholic. It is positively bad when it depends upon disease of the spinal cord.

TREATMENT. In a recent neuritis, we may apply counter-irritation over the nerve at the lower and inner part of the anterior triangle of the neck. Electricity is not considered to be of much avail in cases of any considerable gravity. If it is to be used, one pole (anode) should be pressed deeply into the structures outside the lower part of the clavicular portion of the sterno-cleido-mastoid muscle, while the other pole is placed upon the sternum at the border of the epigastrium, or over the diaphragm on the same side as the electrode. Relieve asphyxia by oxygen.

THERAPEUTICS. The same as in other forms of neuritis.

THE LOWER PLEXUS.

ANATOMY. Owing to their position, the common accidents of life do not so readily compromise the nerves of the lower extremity, and, with the exception of sciatica, they do not seem to be so liable to primary forms of neuritis. The other nerves of the plexus are more apt to be involved in a general neuritis, ascending from an infection of one of the nerves lower down. They are liable to injury by diseases of the vertebræ, and of the meninges, abscesses and inflammations of the pelvic viscera, and are peculiarly vulnerable to the pressure and toxic conditions dependent upon gestation and parturition. Paralysis has resulted from section of nerves in operations on the abdomen.

The muscles of the abdomen and legs are innervated from the lumbar and sacral plexuses. The lumbar (L, 1, 2, 3 and half of the 4th) supplies the skin of the lower part of the abdomen, the front and sides of the thigh,

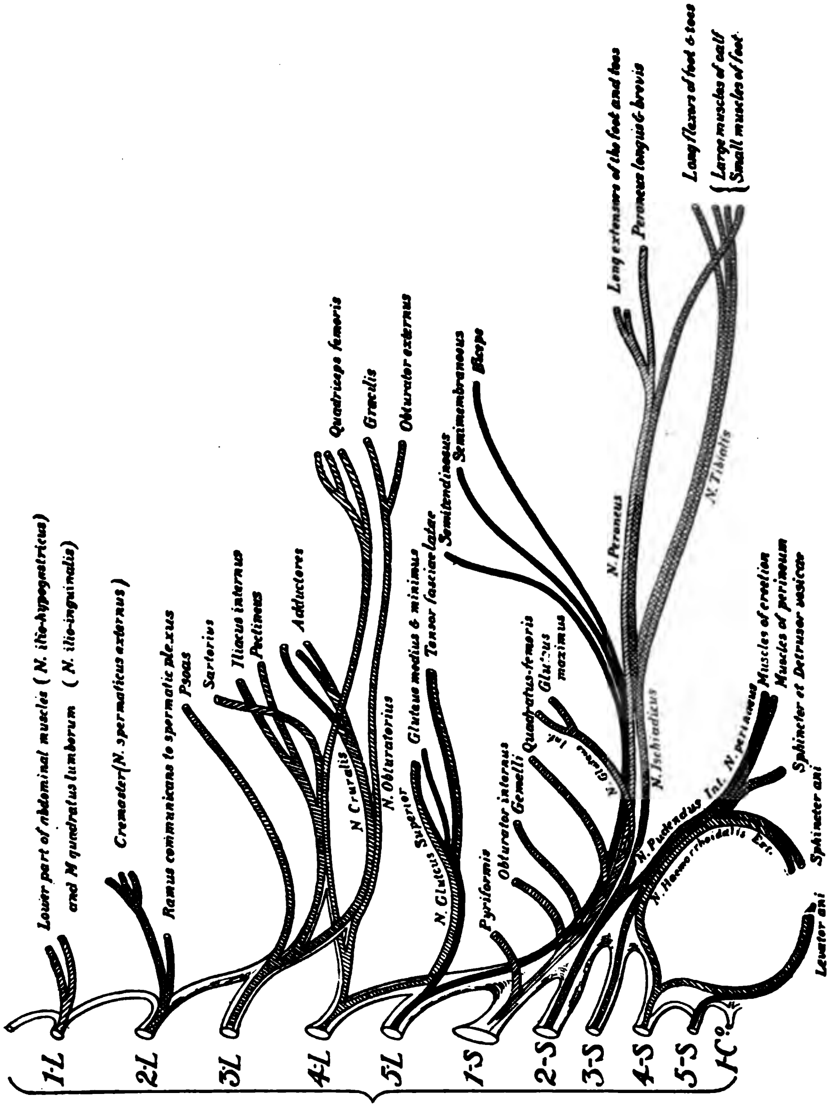


FIGURE 58. THE LUMBO SACRAL PLEXUS. (From Kocher.)

and the inner side of the lower leg, and inner side of the foot. It innervates the cremasteric muscle, those which flex and adduct the hip-joint, and those which extend the knee. The nerves to the thigh are the obturator, and the anterior crural, the sacral plexus (lower half of the 4th, L, 5, L, and all the sacral), only the upper four having influence over the leg. This plexus innervates the extensors and rotators of the hip, flexors of the knee, all the muscles which move the foot, the skin of the gluteal region, of the back of the thigh, outer side and back of the lower leg, and most of the foot (all except a strip, on the inner side). These nerves are chiefly branches to the outward rotators of the hip and the gluteal nerve, the small sciatic, and the great sciatic.

SYMPTOMATOLOGY OF LOWER PLEXUS PALSIES. Symptoms of injury to the plexus may be confined to such symptoms as would flow from an injury to the roots. The pain, in a neuritis of the plexus, may extend along the course of the ilio-hypogastric, ilio-inguinal and genito-crural to the lower part of the abdomen, and the groin.

OBTURATOR. This nerve is rarely affected alone, but pressure may be so localized as to produce it.

SYMPTOMATOLOGY. The thigh cannot be adducted, and so one leg cannot be crossed over the other, and external and internal rotation is weakened. Sensory changes occur in the upper-middle part of the thigh.

ANTERIOR CRURAL. This may be affected either above, or below Poupart's ligament. In the first instance the iliacus is weakened, while it escapes if the lesion is below this point. It may be irritated by a growth near the spine, or paralyzed by it. This nerve is frequently affected in parturition. If the iliacus is involved, flexion of the thigh upon the body is weakened, but if the lesion is issitative only, there is pain in the region of its distribution. In a paralysis below this muscle, the extension of the knee is compromised, and the loss is most conspicuous in walking, since the leg cannot be advanced satisfactorily. Mounting stairs is almost impossible. The usual cause of injury is in fracture of the thigh, and the quadriceps extensor is the muscle almost exclusively paralyzed. Sensory loss or change is from the groin to the inner side of the foot, excepting the buttock and a narrow strip down the back of the thigh, which, below the knee, spreads over the outer side of the calf, and embraces all of the foot, except on the inner margin. Gower says it also goes to the adjacent sides of the first and second toes.

SUPERIOR GLUTEAL (L, 4-5). Rarely affected in an isolated neuritis, and goes to the gluteus medius and minimus, and the tensor vaginæ femoris. It lies between the two plexuses in origin.

SYMPTOMATOLOGY. Its paralysis interferes with abduction, outward rotation, and circumduction of the thigh.

SACRAL PLEXUS. This is rarely affected in a primary way, and sensory diseases practically mean sciatica, from which, secondarily, the plexus may be involved in a neuritis, and in a motor form we are dealing with disturbances of the peroneal nerve. It may, as a whole, be damaged by growths and inflammations in the pelvis, and bruised or compressed in parturition, but the paralysis is due to a combination of an anemic condition and pressure or bruising. Tension from abnormal strains occasionally is the cause.

SCIATIC NERVE (4 L-3 S.). This nerve may be injured from compression, laceration, by growths, or by fractures, and is subject to neuromata. The external popliteal is as exposed as the musculo-spiral, and suffers in like measure from injury, and often is the seat of spontaneous neuritis. It may also be injured by any fracture of the bones. It has been considered subject to neuralgia, but investigation will generally reveal changes in the nerve.

SYMPTOMATOLOGY OF PARALYSIS OF THE WHOLE NERVE If the injury is near the sciatic notch, there is a paralysis of the flexors of the hip, and of all the muscles below the knee, but walking is still possible, from the possibility of raising the foot by over-flexion of the hip. The leg is thrown forward as a whole, the knee being kept in extension by the anterior crural. The result is a peg-leg, such as we get in hemiplegia, the only difference being that in hemiplegia the leg is spastic. Often the lesion is below the middle of the thigh, and then the flexors of the leg escape. It is not so disabling as a lesion of the anterior crural, the loss of power is usually partial, and the knee-jerk is preserved. Sensory losses from a lesion below the origin of the small sciatic, are in the outer half of the leg, the greater part of the dorsum of the foot, and all the sole, but it is possible that the leg may escape, owing to anastomosis with other sensory nerves. Atrophy is marked in the back of the thigh, and below the knee. It is possible that there may be perforating ulcer of the foot, especially of the ball of the great toe.

PARALYSIS OF THE BRANCHES.

EXTERNAL POPLITEAL (PERONEAL). This nerve is especially liable to injury in the popliteal space, and as it turns about the outside of the fibula. It is sometimes injured in jumping (on hard ground especially), and from taking a false step. Its injury paralyzes the tibialis anticus, the long extensor of the toes, the peronei, and extensor brevis digitorum.

SYMPTOMATOLOGY. There is loss of the power of flexion of the ankle, and

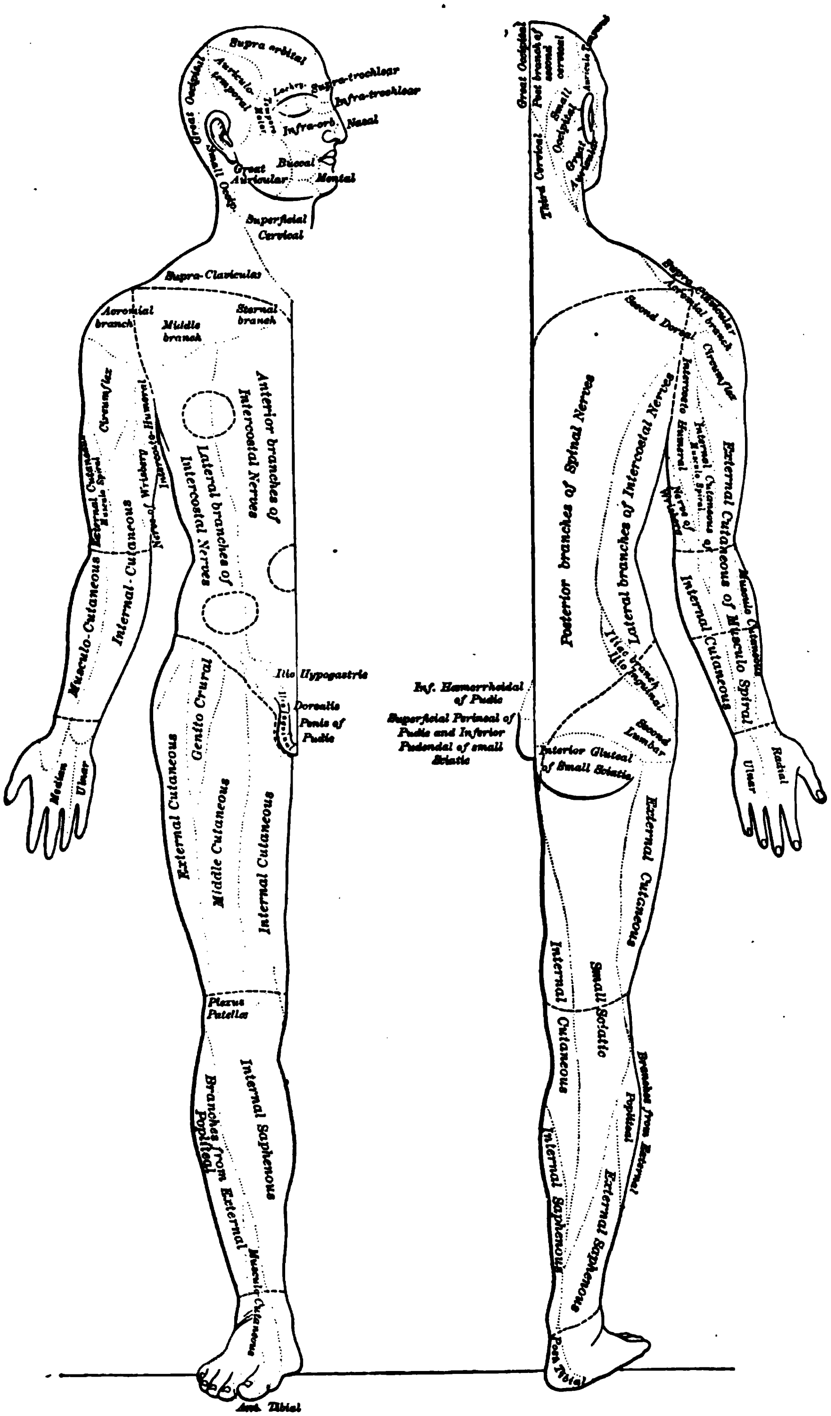


FIGURE 59. THE DISTRIBUTION OF SENSORY NERVES IN THE SKIN. (After Flower.)

The areas of the skin supplied by the cutaneous nerves are shown in finely dotted outline. The circles on the trunk show areas occasionally anesthetic in hysteria. The lines across the limbs at ankle, knee, and thigh, wrist, elbow, and shoulder show the upper limits of anesthesia in multiple neuritis of varying degree of severity. (Starr. Courtesy of Lea & Febiger.)

of extending the first phalanx of the toes. This is objectively striking in the production of foot-drop and toe-drop, and from the fact that the toes drag upon the ground in walking. From the contraction of the unopposed interossii, the toes curl in upon the sole of the foot, and still further embarrass the sufferer. Contraction of the tendo-Achilles is a common result, and causes the foot to assume the equino-varus position. The paralysis is a flaccid one, and there is the reaction of degeneration. Sensory loss is an anesthesia of the outer half of the front of the leg, and of the dorsum of the foot. The fullness of the muscles on the anterior and outer aspect of the leg is lost.

INTERNAL POPLITEAL. This is analogous to the median and ulnar in the upper limb, while the external popliteal resembles the musculo-spiral in the course and symptoms of its injury. It is distributed to the popliteus, the calf muscles, tibialis posticus, the long flexors of the toes, as well as to the muscles of the sole. It divides into the internal and external plantar, which may be separately diseased.

SYMPTOMOATOLOGY. There is a loss of the power of inward rotation of the flexed leg, if the site of the disease is high enough to involve the branch to the popliteus, and there is a loss of the power of extending the ankle-joint. The result is talipes calcaneus. Sensory loss is variable in amount, but typically it is on the outer part of the back of the leg, and on the sole.

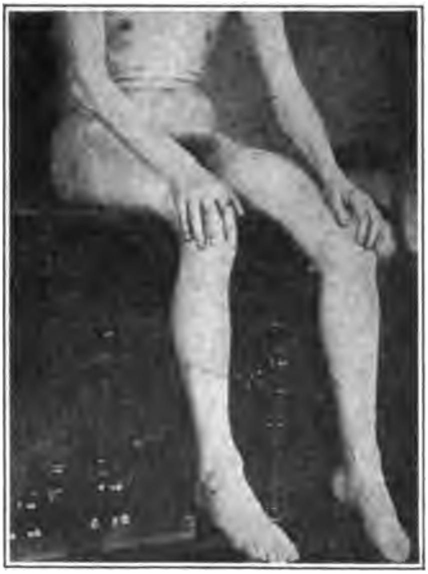


FIGURE 60. DROP-FOOT. (From Oppenheim.) Courtesy of J. B. Lippincott.

INTERNAL PLANTAR. Paralyzes the short flexor of the toes, the plantar muscles of the great toe (except the adductor), and the two inner lumbricales.

SYMPTOMATOLOGY. The toes cannot be flexed at the second phalanax, and so we find "hammer-toe," see Fig. 67, and clumsiness in walking. Sensory loss is anesthesia of the inner part of the sole, and the plantar surface of the three inner toes and half of the fourth toe.

EXTERNAL PLANTAR. This is distributed to the flexor accessorius, the muscles of the little toe, and all the interossii, the two outer lumbricales, and the adductor of the great toe.

SYMPTOMATOLOGY. This paralysis flexes the last two joints of the toes, and extends the others, from the contraction of the opponents of the interossii. This position of the toes makes walking almost impossible. Sensory loss is an anesthesia of the outer half of the sole, the little toe, and half of the fourth toe.

Metatarsalgia (Morton's painful toe) results from a pressure neuralgia, or neuritis of one of the digital branches of the plantar nerve It is compressed between the heads of the metatarsal bones, probably as a result of a congenital malposition of the nerve.

TREATMENT of all these conditions is postural, electrical and symptomatic.

NERVE SUPPLY TO MUSCLES OF THE PELVIS AND LOWER EXTREMITIES.

The Gluteus maximus (inferior gluteal nerve) extends the thigh, and with flexed limbs, the trunk upon the thigh. It slightly rotates the thigh outward. If paralyzed the pelvis is supported when the thighs are in strong contraction, and so the pelvis is tilted forward. The Medius (superior gluteal) is more of an abductor of the thigh than anything else; it rotates it inward, and with a flexed leg it draws the trunk to one side. Gluteus maximus (sup. gluteal) has a similar action. When these muscles are paralyzed the leg cannot be extended, nor abducted; there is great difficulty in climbing, and there is a wabbling gait, because the pelvis is unsupported, and its motility is exaggerated.

The Pyriformis, the Gemelli, External obturators, and Quadratus femoris (all sacral plexus) rotate the upper thigh outward, so if paralyzed the leg is continuously turned inward.

The Ilio-psoas (2d and 3d lumbar), Psoas (anterior crural) flex the leg at the hip joint, and turn the thigh slightly outward. When paralyzed the thigh cannot be flexed when lying down, and there is difficulty in rising from a horizontal position. When both flexors are paralyzed walking is impossible.

The Pectineus, Adductores, and Gracilis (obturator nerve to all) adduct the thigh, the Pectineus flexing it at the same time, all three causing a slight rotation outward. If paralyzed the leg cannot be adducted, and it will turn outward from the overweight of the abductors.

The Quadriceps femoris (ant. crural) extends the lower leg. In paralysis of it standing may be possible, but walking is impossible, as even while sitting the patient cannot extend the leg on the thigh.

The Vastus internus (ant. crural) extends the leg. If paralyzed alone it may allow the Vastus externus to produce a luxation of the patella outward.

Sartorius (ant. crural) causes flexion of the hip and knee joints, and at the same time abducts the thigh, and rotates it outward incompletely. If paralyzed flexion is imperfect. The Gracilis (ant. crural) flexes the lower leg slightly, adducts the leg more, and rotates it inward.

The Biceps (int. popliteal) is an outward rotator. The Semi-tendinosus and Semi-membranosus (both int. popliteal) are inward rotators. They are all flexors of the lower leg, and extensors of the hip. If paralyzed the leg cannot be flexed and walking, dancing, and running are impossible, the action of the Quadriceps is unopposed, and the thigh is flexed to excess, and trunk is moved backward.

The Popliteus (int. popliteal) rotates the outer leg when flexed inward, and flexes itself very slightly.

The Gastrocnemius, Plantaris (both int. popliteal), and Soleus (int. popliteal and post. tibial.) promote plantar flexion and adduction of the foot. The foot will also be turned so that the dorsal surface looks outward. In simple plantar flexion the Peroneus longus is also called into action, which is more forcible with the extended knee. In paralysis of these muscles, flexion of the foot is almost impossible, the patient cannot raise himself upon his toes, and walking is difficult. Deformities ensue from the action of the unparalyzed muscles.

The Peroneus longus (musculo-cutaneous branch of the ext. poplitael) is essentially an abductor. It depresses the inner, and elevates the outer border of the foot, and with abduction of the front end. In paralysis of this muscle extension of the foot takes place with adduction. Walking is fatiguing, for the plantar arch is dropped, standing on the toes is not possible, or is uncertain. Pressure on the nerves ot the sole of the foot while walking produces paresthesias and pain.

The Tibialis anticus, Extensor longus digitorum, and Extensor hallucis (ant. tibial.) produce extension of the foot. In paralysis there is no extension of the foot, it cannot be raised, and hangs flaccid as soon as it is raised from the floor. In walking the toes catch on the floor, and to prevent this in walking the leg is flexed at both the hip and the knee. In chronic paralyses contraction of the flexors results, and "pes-equinus" or "equino-varus" is produced. If the Tibialis anticus only is paralyzed, extension of the foot is combined with abduction.

The adductor, flexor brevis, and Adductor hallucis (int. plantar nerve) flex the first phalanx of the great toe, and extend the second. The abductor, and inner head of the flexor brevis contract, in contraction of the foot, preparatory to pushing it from the floor.

The Extensors of the toes (ant. tibial nerve) being paralyzed the Interossei become chronically contracted. The first phalanges are flexed, the last are extended, producing an abnormal position of the toes.

The Interossei (ext. plantar nerve) if paralyzed produces an over-extension of the first phalanges, the head subluxated, the second and third flexed, producing "Claw-foot." Walking is not impeded, but it is painful.

LESIONS OF THE CAUDA EQUINA AND CONUS MEDULLARIS.

Since the cord ends in a conical point, termed the conus terminale, at the level of the second lumbar vertebra, the fibres emerging at the lower levels of the spine must pursue a somewhat extended intra-spinal course. They take their rise from the lower portion of the cord, in close proximity to one another and fall like a coarse fringe which is gathered into a loose bundle by the membranes (theca) which extend as a mantle as far as the second sacral vertebra where they resolve themselves into a cord (filium terminale), which is attached to the fifth sacral vertebra or the upper surface of the coccyx, and so anchors the cord at its lower extremity. The different spinal roots penetrate the theca when they come to a point in their descent opposite to their respective foraminæ between the vertebræ.

From these anatomical facts it follows that cauda equina disease will be marked by no segmental symptoms, but by motor root symptoms if the anterior fibres are affected, and by sensory root symptoms if the posterior roots are affected.

ETIOLOGY. Primary disease is rare, tumor excepted, since all lesions are generally secondary to disease of the spinal meninges, or to disease or injury of the lower vertebræ or sacrum. Fracture of the spine below the first lumbar, bullet wounds, and hemorrhages from injuries, too slight to actually cause fracture are common causes. The meninges are liable to infection in this locality from syphilis, carcinoma, and tubercle, and the latter may produce the damage by the formation of abscess. The lesions are usually partial.

SYMPTOMATOLOGY. In partial lesions the inner fibres, i. e., those which are sacral and coccygeal are affected, and the outer ones only in more complete lesions. If the lesion is a complete one there are paralysis and atrophy of all the muscles of the lower extremities, anesthesia of the buttocks and perineum, and genitals. The knee-jerk and Achilles-jerk are lost, the plantar and anal reflexes also; there are paralysis of the bladder and rectum, and loss of sexual power. The first lumbar generally escapes, and in this case the sensation is normal in the inguinal region, and the anterior part of each thigh. If the first, second, and third lumbar escape, the anterior crural and obturator nerves retain their function, and only the glutei and the ham-string muscles are paralyzed above the knee, but all below the knee are of course affected. The anus and bladder are paralyzed, but the knee-jerk and the plantar reflex are normal. The anesthesia will follow the distinctive distribution. As the site of the lesion descends, the muscular paralyses and the anesthesias will of course be correspondingly reduced, until its situation is such that the second sacral is spared; then we get a characteristic anesthesia, which is of a saddle-shaped portion of the skin, including the anus, perineum, the posterior aspect of the scrotum and penis, a small strip of skin running from the perineum along the posterior-internal aspect of the thigh, but producing no anesthesia below the knee. The urethral and vesical mucous membranes are anesthetic, the bladder and rectum are paralyzed, the anal reflex is lost; there is no disturbance of the knee-jerk, or the plantar reflex. Lesions of the lower roots of the cauda equina give a more limited palsy as the lesion is lower and lower in height. First the genital apparatus will escape, next the bladder and rectum, until we come to lesions affecting only coccygeal nerves, when there will be a cutaneous palsy of the anus and perineum, but the sphincter will be intact.

DIAGNOSIS. This is between the involvement of the whole cauda equina, and of the segments of the cord from which these nerves would arise. This is very essential, for a lesion of the conus is beyond operation, while a lesion of the cauda may be successfully operated. Anatomically the conus is that portion of the cord which is covered by the second lumbar vertebra, at the lower border of which vertebra the filium terminale begins. Physiologically it has been considered to contain the centers for micturition, defecation, erection, ejaculation, and for the anal reflex, but Mueller states that these reflex centers lie in the pelvic sympathetic ganglia, and that the volitional centers alone are located in the ganglion cells of the conus. On this account a lesion will affect the external sphincters only. There is no difference in the distribution of the palsy, but it is lower neuron in type if the lesion is in the cauda equina; that is, there is a muscular palsy, atrophy, the reaction of degeneration, absence of knee-jerk, and normal bladder reflex, vesical and anal reflexes, with constant dribbling of urine. Lesions of the cauda are apt to be extremely painful, the pain radiating into the legs, following the sciatic nerves especially. If the lesion causing muscular paralyses and anesthesia in a similar area is located in the spinal cord above the segments of origin of these nerves, instead of in the cauda equina, we shall have an anesthesia, and a muscular palsy, but no atrophy, no reaction of degeneration, the knee-jerk will be increased, there will be ankle clonus, and Babinski's reflex, and we shall have reflex urinary incontinence, there will be retention of urine at first, and as soon as the lower centers recover from the shock of the initial lesion, the bladder will empty itself when full, without the knowledge of the patient. If the lesion is in the segments controlling these actions, it cannot be distinguished with certainty from a lesion of the same fibres in the cauda. If the conus is affected, we shall have the muscles of the lower extremities intact, anesthesia in the saddle-shaped area referred to, and loss of sexual power; also Babinski's reflex and a lack of rectal control, but the onset of the motor palsy will be very rapid, and the atrophy especially so, with the result that there is a great tendency to bedsores. There will be no pain, but partial sensory lesions.

PROGNOSIS AND TREATMENT. The prognosis of a lesion of the cauda is better than one involving the cord, since the nerve fibres which make up the cauda are capable of regeneration, while the tissues of the cord itself are not replaced after degeneration. Syphilitic exudates are more tolerable in this position, and hemorrhages are more liable to absorption. If the compression is from new growth of any kind, it is in a position where removal is among the possibilities, and whatever disease or injury may have been suffered by the bones in this locality can be detected by X-ray, and is not beyond the reach of surgery.

THERAPEUTICS. As has been indicated, the treatment for most conditions affecting the cauda equina is surgical, and if it has been successful, the results of the compression of the nerve roots should be combatted by massage, electricity, baths, and such remedies as Hypericum, Arnica, Ranunculus, Bellis perennis, Plumbum, Rhus, Causticum, and Phosphorus; the first two in the earlier stages of recovery, while Phosphorus should be considered in the last attempt to stimulate a degenerated nerve. In case of hemorrhage we may promote absorption by Hepar sulphur, Graphites or Silicea, while if the case is syphilitic specific treatment is much more efficacious.

CHAPTER IV.

DISEASES OF THE SPINAL CORD.

The spinal cord is subject to malformations from a failure of developement in the arches of the vertebræ, or of the medullary folds, or there may be faults in the developement of the cord itself.

Spina bifida is the most common malformation, and it is due to a defect in the developement of the vertebral arches, and at times of the medullary folds as well. It is the projection in the posterior median line of a sac, which may be spherical or pedunculated, containing cerebrospinal fluid, from one to six inches in diameter. It commonly occurs in the lumbo-sacral region since the arches in this locality are the last to solidify. If the sac is composed of the membranes alone, it is termed a Meningocele, if it also contains some amount of the cord, it is a Meningomyelocele, while if it is of cord-tissue alone, it is a Myelocele. The sac may be covered with membranes and skin, or with membrane only, in which latter case the outlook for life is not as favorable. In all cases the lower part of the cord is apt to be rudimentary.

Two-thirds of all cases of spina bifida are Meningocele. In these cases there is to some degree paralysis of the lower limbs, and atrophy of all the muscles to the lower extremities may take place. On account of this the knee-jerks are absent, and a talipes of the varus or equino-varus type occurs. The sac is apt to ulcerate, there is anesthesia, and bedsores may develope. The bladder and rectum are affected, and contractures of the lower limbs are often a feature of the disability. Surgery is the only treatment and the evacuation of the cyst followed by compression is not advised, but the injection of Morton's fluid, followed by keeping the child upon its back has been followed by success in some cases. Morton's fluid is composed of Iodine grs. 10, Potassium iodide grs. 30, Glycerine 2 ounces. Inject 1 dram on the lateral aspect of the cyst (Dana). The operation should not be done under the age of two or three months, and not after severe nervous symptoms have existed for more than a year. In these deferred cases palliative treatment of an orthopedic kind is the only reasonable course. These children do not usually live more than three months to a year, although with a small tumor some have survived to adult life.

Meningomyelocele is rare. In these cases the sac contains part of the cord, and the spinal nerves emerge from the sides of the sac and re-enter the spinal canal. No operation can be done in these cases, and they do not survive.

Myelocele is a protrusion of the nervous tissue, on account of the failure of both the arches and the medullary folds to coalesce. It is not operable and is fatal.

Craniorrhachischisis is the condition where the entire medullary groove has failed to close up, and therefore the cranial vault, and the vertebral canal lie open, and a mass of vascular membranes are the only representation of the brain and spinal cord.

Rhachischisis is the condition when the spinal canal alone is open. Atelomyelia is a defect arising from the absence of some definite segment from the cord. Diastematomyelia is a bifurcation of the cord. Diplomyelia is a double cord. Amyelia is the absence of a spinal cord, and exists only in cases where no brain has developed, but the absence of developement of the brain does not ensure the absence of a spinal cord. Micromyelia designates a cord smaller than normal, or shorter than normal. It is not a rare condition, and usually is true of a part only of the cord. Heteropia of gray-matter is a term indicating that nodules of gray-matter are found scattered among the white tracts of the cord. This condition has been found in the brain also. Van Gieson states that this condition is very rare, and that as a rule it is artificially produced by post-mortem handling of the tissues.

HYPEREMIA AND ANEMIA OF THE SPINAL CORD.

To anemia and hyperemia of the spinal cord have been attributed a long array of peripheral symptoms, the etiology of which has very seriously been called in question. The arteries of the spinal cord have such a tortuous course, that the arterial pressure in the spinal cord is uninfluenced by fluctuations in the cardiac pressure, and the veins bear such a small proportion to the peripheral venous circulation into which they empty, that they are very little affected by a blocking of circulation in the thorax. They are thus immune from the ordinary variations in vascular equilibrium, but from this very fact would be very slow in returning to the normal if congestion did occur. Functional activity is always associated by vascular dilatation in the functionating area, and the same is probably true of the cord. Actual hemorrhage in the cord has been produced by excessive coitus, extravasations have been found in cases of strychnine poisoning, hydrophobia, tetanus, and it apparently results from excessive muscular exertion, and suppression of the menses. It will then be seen that congestion is a possibility, but that the instances where it has been found are where the functional activity would specifically pertain to spinal centers, or when the local circulation had been suddenly and specifically increased. Chronic hyperemia has been adduced as the cause of the paresthesias and weaknesses of the lower limbs which are encountered at times as a transient symptom, and it has been considered that when the vitality of a person was low, a prolonged dorsal posture might produce such a condition in the cord. The symptoms, in view of the anatomical facts, are to be ascribed with much more probability to a functional disturbance in the cells of the cord, or to affections of the peripheral end-organs.

SYMPTOMATOLOGY. The symptoms of acute spinal-cord hyperemia are the milder phenomena of a myelitis, viz., some loss of motor power, paresthesias, numbnesses, creepings, pains in the loins and down the legs, and possibly some weakness of the sphincters. The symptoms attributed to chronic hyperemia are those which have been attributed to neurasthenia, and are probably so in fact. Patients suffer from muscular weakness, pains of a neuralgic type, paresthesias, tenderness over the bony prominences, especially in the cervical, upper dorsal, and coccygeal region. The coccyx may be the seat of spontaneous pain. The functions may be disturbed, and they are subject to palpitation, dyspepsia, and constipation. There is nausea and headache, and the condition may last for two or three years, and they may become bed-ridden.

TREATMENT. Quiet in a horizontal position on the side or face. Ice upon the spine, and counter-irritation. Extreme pain may demand analgesics. Aconite, Veratrum vir. and Gelsemium are the proper initial remedies; those recommended for myelitis should be used later.

Even less is known about spinal anemia than is known about hyperemia. Anemia may be produced by any local condition which will prevent the perfection of the arterial supply. In cases of general anemia, the spinal cord rulably must suffer, along with the other structures of the body, but as a clinical fact, in some cases of fatal anemia the spinal cord has been found entirely free from such changes as would be expected. Atheroma of the vessels is less common in the cord than in the brain, and that anemia is produced by spasms of the arteries of the cord is difficult of proof, and not very probable. Many symptoms have been attributed to this vascular spasm, such as sudden loss of power in the legs; pins and needles sensations in the arms and legs, and the cramps and rigidities with which people awaken in the night. In all these cases it is much more probable that they are caused by functional changes in the cells of the cord, or intoxications of the peripheral end-organs, for the reasons given in describing the circulation of the cord. It has been customary to associate with spinal anemia a class of symptoms characterized by pains in the back, of the nature of spinal irritation, weakness of the legs amounting to a paraplegia, a type of symptom that has been called spinal concussion, but it is impossible at the present time to say that a spinal anemia underlies and causes this condition.

TREATMENT. Remedies for neurasthenic states are applicable.

SPINAL HEMORRHAGE. SPINAL APOPLEXY.

Spinal apoplexy may result from hemorrhage in the cord itself (hematomyelia), which is rare as a primary disease, or from a hemorrhage into the spinal canal (hematorrhachis), and may be subdural, or epidural, which is not rare, that outside the dura being the most common.

Hematomyelia. Hemorrhage into the substance of the cord. While hemorrhage into the cord is rare as a primary condition, it is secondary to so many processes that it is fairly common.

DIFFERENTIAL DIAGNOSIS. The diagnosis is from Spinal Meningeal Hemorrhage, Acute Myelitis, Acute Anterior Poliomyelitis, Syringomyelia.

ETIOLOGY. It may occur at any age, and to any sex, but most cases occur in males in early adult life, 20-40, on account of the fact that many cases come from over-exertion, exertion under unusual conditions, Caisson disease for instance; from excessive sexual indulgence, one case is reported. It may occur in the new-born, from instrumental violence, or from the vascular strain produced by convulsions at any age. It may be caused by pathological blood-states, like purpura, or in anemias, or from vascular changes incident to old age, syphilis, or the acute infections. It may be secondary to disease-processes in the neighboring structures, like tuberculosis and cancer; it may be the result, or the cause of acute myelitis, or may be due to new-growth in the cord, like syringomyelia. It has been known to follow falls, the victim striking at times on the buttocks, and in some instances landing on the feet.

PATHOLOGY. It is rarer than cerebral hemorrhage, because the arteries are long and tortuous, they are not subject to miliary aneurisms, nor as frequently to degenerative diseases, and the tissues afford a firmer support than those of the brain. Owing to their higher pressure, the ones affected are the central ones to the gray matter. The hemorrhage tends to extend vertically and to confine itself to the gray matter, but it may appear in scattered areas, and there may be numerous capillary hemorrhages. The hemorrhage may involve the entire section of gray matter, or only one-half of it, or only an anterior or a posterior horn alone. The longest vertical extension appears in the cases of central hemorrhage. As a final condition the hemorrhage terminates in a cyst, or a mass of degenerated material which may form the focus of a myelitis.

SYMPTOMATOLOGY. Pain in the back, without premonitory signs, is the usual initial symptom followed in a few minutes by paralysis and numbness which attains completeness a little later, generally with no loss of consciousness. The paralysis is a flaccid one at first, and the sphincters are paralyzed. If the lesion is a complete transverse one, the paralysis will remain flaccid, as will also be the case if the lesion is in the lumbar enlargement. This is a common localization. If it is in the cervical enlargement, the lower extremities will become spastic, and the reflexes will be exaggerated, and if in the lower part, there will be contraction of the pupil, and narrowing of the palpebral fissure. In all cases there will be atrophy of the tissues which are innervated from the areas directly involved, and there may be intense pain in parts just above, on account of the irritation of the nerve-roots from the hemorrhage. The typical sensory symptom is a dissociated anesthesia where pain and temperature are poorly appreciated, while tactile sense is preserved. Brown-Sequard's unilateral syndrome may be found. The temperature is not usually disturbed, unless a myelitis is set up about the clot. If this is the case there will be a secondary extension of the paralysis proportionate to the amount of this new process. Cystitis and bedsores are liable to occur.

Diagnosis. This rests upon the abrupt appearance of a paraplegia, without premonitory symptoms, without pain or with a moderate degree of it. The paralysis is marked by atrophy to some degree, and the dissociated anesthesia is characteristic.

Differential Diagnosis. Spinal Meningeal hemorrhage is slower in the onset of the paralysis, as a rule; the pain is much greater; it is much more extensive and radiates into the limbs; spasms in the muscles of the back are a prominent feature; there is no dissociation of sensation.

Acute myelitis and softening come on more gradually, the paralysis is more extensive, but the peculiar sensory disturbance is lacking. There is an apoplectiform type of myelitis which cannot be distinguished.

Acute anterior poliomyelitis begins with a fever, the paralysis is almost universally motor, as there are no sensory changes unless the posterior columns are invaded, and the bladder and rectum are not paralyzed.

Syringomyelia has a slow onset, and while the sensory changes are the same, the symptoms of hematomyelia tend to improve, except in the acutely fatal cases, while syringomyelia tends to show an increase in disability.

Prognosis. Death is rarely immediate, but is usually caused by the exhausting effects of cystitis and bedsores. Respiratory paralysis in the acute stage is a possibility. Signs of recovery are often seen in the first week, the paralysis diminishes, and the sensory changes are not so evident. On the whole it may be predicted that the danger to life is moderate, that a large degree of improvement may be expected, but that perfect restoration to health is not to be expected.

Treatment. Rest in bed, on the side or face, with ice to the spine.

Counter-irritation is of dubious value on account of the lowered resistance of the skin, and the danger of ulceration. The later treatment is like that of myelitis: massage, warm baths, educational exercises.

Remedies at first are of doubtful utility, except to relieve the pain if prominent. Aconite may be of use from the first; Gelsemium seems the remedy most applicable to the whole condition. Phosphorus and Causticum may be of service in the chronic stage. In the purpuric form Hamamelis, Arsenicum, Phosphorus and the serpent poisons. The old school advise the mineral acids, and Adrenalin.

SPINAL MENINGEAL HEMORRHAGE.

Spinal Meningeal Hemorrhage (Hematorrhachis) is a common accident, and is more apt to occur without than within the dura. It may occur at any age from infancy to senility, but is most common in adult males, from the fact that exertion and traumatism are the most common causes.

Differential Diagnosis. Hematomyelia, Meningitis and Meningomyelitis, Tetanus.

Etiology. In some cases of muscular overstrain the arteries of the meninges rupture, and there is an extravasation of blood into the subdural space which may fill it for the space of two or three vertebræ, or to its whole extent. I saw such a result in a feeble lady after osteopathic manipulations. The convulsions of tetanus and eclampsia have produced such a hemorrhage, purpura has been responsible, and forcible delivery has caused it in the infant. All these hemorrhages are of especial importance, since they produce compression of the cord. The extradural hemorrhages are generally too small to do this. They are the result of the rupture of one of the veins in the plexus between the spine and the dura, caused often by disease of contiguous parts, convulsions, and sometimes birth-injuries. When an aortic or vertebral aneurism necroses the vertebræ, and protrudes into the extradural space it may exert very disastrous pressure upon the cord. I have seen a severe compression myelitis from this cause. Fracture dislocations of the spine produce both extradural and intradural hemorrhages at the same time, and if the cord is not crushed, they still furnish the large percentage of the meningeal spinal hemorrhages.

Symptomatology. The symptoms will be mainly produced by pressure upon the cord, or from a reactive meningitis. If the clot is small and extradural there may be no distinctive symptoms. If it is large, there will be pain in the back over an extensive area, spasms in the muscles of the back, numbness extending over the lower part of the back, and down the legs, and some degree of paralysis. This takes only from a few minutes to a few hours to develope. The pain and paresthesias and anesthesias along the nerves from the affected segments are very pronounced, and the paralysis and loss of sensation below the lesions are later developements. There is retention of urine. In three or four days there is improvement, unless myelitis or meningitis is developed. The tendon reflexes are diminished at first, and afterward increased unless the compression has been severe enough to destroy a reflex arc.

PROGNOSIS. From such a varied etiology, it may be seen that there is abundant opportunity for fatality, but if the patient survives the first three or four days, there is large probability of his recovery. Cystitis and bedsores may kill him from exhaustion, if the compression myelitis is severe, or he may die of respiratory paralysis, if the hemorrhagic area involves the medulla. With good care, cases left with very severe spastic paraplegia may recover a very tolerable degree of efficiency.

DIAGNOSIS. The history of childbirth, or of injury, or the discovery of some disorder of the blood, or other pathological condition may direct us to the diagnosis. Severe pain over much of the spinal region, with cramps and paresthesias extending down the legs, followed in a few hours by paralytic symptoms, all indicating a form of compression myelitis, are very suggestive. Hematomyelia has less pain, but more paralysis, atrophy, a dissociation of sensory loss, and is more abrupt in onset, unless the hemorrhage was from a fracture dislocation of the spine. In tetanus the symptoms develope slowly, and trismus is present.

TREATMENT. Absolute rest on the side or face, with ice to the spine, and the pain may be so severe as to demand analgesics liberally. Counter-irritation, venesection or leeching are not generally endorsed, although advised by some. In case of fracture-dislocation surgery is sometimes indicated, and especially so if the case is seen within an hour or two of the injury. Where the diagnosis is fairly certain, it might be advisable to try the effect of lumbar puncture. Remedies and subsequent treatment are as in Hematomyelia.

CAISSON DISEASE. DIVERS' PARALYSIS. THE BENDS.

DEFINITION. This is the term applied to the combination of cerebral and spinal symptoms produced in caisson-workers and divers, from a too rapid return to the normal atmospheric pressure, after working in air under high pressure.

ETIOLOGY. It is caused by remaining for too long a time in an atmospheric pressure too high to allow the normal osmosis of inspired gases.

It differs with various people, it depends largely on the length of time to which a person is subjected to these conditions, and on the degree of compression. Workers become habituated to increased pressure, but dissipation and exhaustion of any kind render them more susceptible. In actual practice, most cases arise from the laborer's desire to leave his work as quickly as possible, and thus he does not remain in the airlock a sufficient time; also because an airlock is a foggy, chilly place.

PATHOLOGY. The gases in the blood are at high pressure, and on reaching an environment at a much lower pressure, osmosis fails, and they circulate in the vessels as bubbles, form air emboli in the capillaries, or rupture their walls. Hill and McLeod have demonstrated the bubbles of gas in the capillaries in a frog's foot, and a bat's wing. These bubbles could be seen, arresting the circulation in the small vessels. By this process lacerations, vacuolizations, and areas of softening are formed, especially in the nervous system, perhaps on account of the tortuosity of the blood-vessels, and perhaps because the tissues give so little support to the vessels. Dana considers that the condition is not the result of a congestion of the viscera, but of the mechanical pressure on the surface of the body. When this pressure is relieved there is an attempt at balance, but the brain and spinal cord, being in bony cases, cannot relieve themselves soon enough to avoid catastrophe.

SYMPTOMATOLOGY. The brain symptoms are rulably less pronounced than those of the spinal cord, where the picture is much like a myelitis. Immediately after coming into the outer air, or shortly after, the person is apt to have headache, vertigo, nausea, and perhaps vomiting. He staggers about, and has ringing in the ears, and may show mental disturbance. The prominent symptoms, however, are pains in the legs, arms not usually being affected. The pains are referred to the joints, without swelling, but with weakness, and they feel heavy and numb. Soon the victim will be paraplegic to some extent, and will have anesthesias and paresthesias of irregular distribution. The sphincters are often affected in the line of retention. These pains in the limbs may be very severe, and so much so as to bend the person over, which gives one of the popular names for the disease, "The Bends."

PROGNOSIS. The disease lasts from a few hours to five or six days and is fatal in from three to five per cent. of the cases. Complete recovery is possible, but most cases have a heightened susceptibility, or some weakness, less often a hemiplegia, or a monoplegia, and some cases are paraplegics. The condition of the severer type of cases is like the result of a myelitis. A few fatal cases die suddenly, with or without delirium, and convulsions.

TREATMENT. Slow decompression is prophylactic, and replacement in an airlock and very gradual decompression is the treatment. For the later treatment, that given in the article on myelitis is appropriate.

SPINAL MENINGITIS. PACHYMENINGITIS SPINALIS.

MENINGITIS affecting the spinal dura is divided into external and internal as in the cranium, but since the spinal dura is not directly applied to the vertebræ as the cerebral dura is to the skull, the condition arises from different causes, and leads to different results. When the internal layer is the seat of inflammation, the other membranes are as commonly implicated as when the disease is located in the cranium, but since the septa of the cord are merely prolongations of the pia, the secondary damage to the cord substance finds no counterpart in the case of the similar disease in the brain.

PACHYMENINGITIS SPINALIS EXTERNA.

(*External Pachymeningitis. Peripachymeningitis and Perimeningitis.*)

DEFINITION. This is a form of inflammation of the dura which is not confined to the external layer of the dura, but involves also the fatty tissues, and the veins which lie between it and the vertebræ. It does not tend to implicate the inner layer of the dura, even when the inflammation is most intense. Under some conditions however all the membranes are involved in a common inflammation, and the cord is constricted by the new-growth. The inflammation is of the chronic type.

SYMPTOMS OF A TYPICAL CASE. After a preliminary disease in some part of the spinal column, the pain in the back increases, and there is a change of sensation in the arms. This is succeeded by twitchings in the muscles, then loss of power and sensation, followed by atrophy, with the reaction of degeneration, and by contractures. Later there may be a spastic paralysis of the legs, and as a final stage this paralysis may become flaccid with disturbance of the sphincters. All these symptoms are developed slowly and intermittently.

DIFFERENTIAL DIAGNOSIS. This is from Multiple Neuritis and Myelitis.

ETIOLOGY. Injuries to the spinal column may cause it, and it has been considered to have resulted from the infection of purulent foci in the immediate neighborhood, carbuncles for instance. The present belief is, however, that it practically always results from osteomyelitis from tuberculosis, and that when near-by foci of pus have been found they are extensions from the spinal canal, and not the opposite. Syphilitic new-growths, and the extensions of cancerous infiltration are occasional causes.

PATHOLOGY. Examination reveals a hyperplastic thickening of the dura to all degrees, from a slight increase, to a sort of nodulated tumor, which may invade the subdural space, or fill it solidly to the vertebræ, or even necrose these bones by its pressure. The nodules may be scattered about, or be aggregated into one mass. The changes are commonly limited to the immediate vicinity of the diseased vertebræ, but in some instances have been found to extend throughout the whole length of the canal. The fat in this space disappears, and the overgrowth often unites the dura and the periosteum of the spinal canal. The surface of the dura is sometimes only reddened, with a little lymph upon its surface, but usually the deposit is purulent, and the amount may be so large that it invades the surrounding structures. It may be sufficient in quantity to compress the cord.

Symptomatology. It will render the symptomatology more evident, if we observe that it mainly arises from the compression and inflammation of the nerve-roots. This is a necessary result, since they pass out of the spinal canal through the pia and the dura, and their coverings are prolongations of these membranes. There is tenderness over the spine, at the vicinity of the inflammation, and shooting or constant pain of quite an intense character in the distribution of the implicated nerves. The pain in the back is sometimes referred to the region of the loins. The skin in the distribution of these nerves is hyperesthetic. The muscles become rigid, and movements of the back are usually restricted and painful. They are the seat of twitchings and spasm. As the compression of the nerves becomes more intense, anesthesia takes the place of hyperesthesia, and muscular twitchings are replaced by paralysis. This is an evidence that degeneration of the nerve-fibres has been produced. When the secretion of pus has been sufficient to compress the cord, we shall have a compression myelitis with a spastic paralysis, and hyperesthesias followed by flaccid paralysis and anesthesias, paralysis of the sphincters, lividity of the skin, and perhaps bedsores. In such acute cases there will be rigors, and fever up to 110°, and profuse sweating. The completeness, or incompleteness, of such results will indicate the degree of compression.

Course and Prognosis. In some cases it runs an almost unnoticed course, and continues for a long time. The primary affection usually determines its outcome, and when it results from caries, with a cure of this condition, there may be a return to health, even after a very decided compression of the cord for some months.

Diagnosis. The causative condition will commonly give rise to the symptoms which would direct one's attention to the diseased locality. Allowing the existence of the symptoms, we should judge that an external pachymeningitis had resulted, if paresthesias, and muscular twitchings, and a wider distribution of the spinal pains were discovered. If the spinal muscles showed a deep edema, we should suspect a purulent meningitis; and collections of pus in the pleura, posterior mediastinum, back of the abdomen, or behind the pharynx, or sudden compression of the cord, would indicate it all the more forcibly.

DIFFERENTIAL DIAGNOSIS. Multiple neuritis differs in these points. In this disease all four limbs are usually involved, while in pachymeningitis the legs are usually free, unless the cord is compressed. The anesthesias of multiple neuritis are on the distal portions of the limbs only, and the trunk is free, while the contrary is the case in pachymeningitis. There is no tenderness of the nerve-trunks in pachymeningitis, and there is evidence of injury to the cord in some cases.

Myelitis has an acute, and not a chronic onset, no pain except a girdle sensation, has early paralysis, bladder and bowel symptoms. Pachymeningitis runs a chronic course, has more pain, spasm and irritation, and the paralysis is a late symptom.

TREATMENT. This is covered primarily when the osteomyelitis has been corrected by the application of orthopedic apparatus. Calcarea carb., Fluoric and Nitric acid, Silicea, and Hepar sulph., in the lower potencies, or in the form of Calcium sulphide, will now have a decided influence upon the necrotic process in the bones, and the absorption of the resulting exudates. If the cause is syphilitic, the treatment should be specific, and if carcinomatous the case is a hopeless one.

PACHYMENINGITIS SPINALIS INTERNA.

DEFINITION. This is a very rare disease, the result of an inflammation, of the chronic type, in the internal layer of the spinal dura. While it may be extensive in its distribution, the majority of the cases are confined to the cervical region, and this variety is termed pachymeningitis cervicalis hypertrophica. It is probably a hemorrhagic condition, and while it generally involves the soft membranes in the inflammation, and also the substance of the cord, it, so far as symptoms result, is a proliferation and a fibrinous thickening of this membrane, with the production of symptoms depending upon the compression of the nerve roots.

THE SYMPTOMS OF A TYPICAL CASE. After a period of indefinite pains in the spine, and stiffness of the neck and back, there is a disturbance of sensation in the forearms, followed by muscular twitchings, then loss of sensation, and finally paralysis of the arms. This paralysis is followed by contracture, in either over-extension or flexion. If the lesion is in the lumbar region, there is the same condition in the legs, with disturbance of the sphincters.

DIFFERENTIAL DIAGNOSIS. This is from Syringomyelia, Diseases of the

Spine, Progressive Muscular Atrophy, Transverse Myelitis, Tumor, and External Pachymeningitis.

AGE. Adults are usually the victims, but sometimes it affects children.

SEX. Males almost always, from causes disclosed by the usual etiology.

ETIOLOGY. Syphilis, alcoholism, traumatism, and exposure have been considered to be causative, but the more frequent employment of lumbar puncture has emphasized the truth of the hypothesis, that it is almost universally syphilitic.

PATHOLOGY. Wherever it is located, the primary process is the rupture of an artery, or a fibrinous exudate from it. The primary layers are therefore of fibrin mixed with blood, while the later ones are more purely fibrinous. The character of the process determines that the layers should be reddish brown at the base, with clearer ones superimposed, which are spotted with the stains of hemorrhages. This fibrinous deposit shows all degrees of hardness up to ossification. When the other membranes become involved in the process, the circulation of the cord is compromised, a marginal myelitis is set up, and as the pia sends septa into the substance of the cord, between the layers of which develope the lymph channels of that structure, there follows a lymphatic congestion in the cord which produces cavities, and the general process may induce a myelitis. Connective tissue may extend along the blood-vessels, and the compression of these vessels will produce areas of softening. Gliomatous overgrowth may also be a result. When the process is purely of the dura, the emergent nerve-trunks are compressed, and they may become atrophied and sclerosed. A compression myelitis may be set up by the pressure of the exudate developed upon this inner layer of the dura. If the soft membranes are also affected, the first result will be a marginal myelitis, while if the substance of the cord undergoes a pathological change, there will be any grade of myelitis, up to a complete transverse section. The distribution of this process is sometimes very extensive, and may extend the whole length of the cord; it generally is confined to a small vertical area in the cervical region, and to the upper, or the lower part of the cervical enlargement. When the cervical region is affected there may be an extension to the medulla and pons, or, on the other hand, the process may be just as closely confined to the lumbar enlargement. The cerebral dura is often affected at the same time.

SYMPTOMATOLOGY. The disease is essentially chronic, and has been divided into three stages, which may be separated by years and months. These are the neuralgic, the paralytic, confined to the arms, and the stage where there are spastic symptoms in the legs. In the first stage there will be pain in the neck, and perhaps in the back of the head, and between the shoulders, and the spinal vertebræ will be tender to percussion. There will be tension and stiffness in the neck, and the head will be carried forward, and motion to either side will be restricted. The pains in the arms will be of an intense neuralgic type, especially along the ulnar and median nerves, and there will be slight muscular stiffness in the arms and hands. These symptoms depend upon the compression of the posterior nerve-roots, and generally last from two to five months; but it may be years before the paralytic stage sets in. This is caused by degeneration of these nerves. The pains continue, but now areas of anesthesia appear upon the members. The muscles affected will be those innervated by the median and ulnar, if the lower part of the cervical enlargement is involved, and the musculo-spiral group, if the disease is in the upper part. The musculo-spiral is most usually the seat of the lesion, and therefore the flexors are most generally impaired. Many authors have taught that there is a characteristic deformity, the so-called "preacher's hand," in which there is over-extension of the wrist joint, extension of the basal, and flexion of the middle and terminal phalanges, but while common, this is not universal, nor diagnostic, depending for its presence upon the site of the lesion. It is, however, a fact that the small muscles of the hand most universally suffer, and that both arms are together, but not equally, affected. The affected muscles atrophy, and display the reaction of degeneration. If the bulb or medulla shares in the changes, there may be a bilateral impairment of hearing, and tachycardia, or the whole symptom complex of bulbar palsy. Oculo-pupillary symptoms sometimes occur, but without any diagnostic peculiarity. If the lesion is confined to the lumbar region there will be pain in the distribution of the nerves of that locality, rigidity of the muscles, and finally talipes. In the third stage the cord shows the signs of compression, and there will be a spastic paraplegia of the lower limbs, with increased reflexes, and disturbance of the sphincters. If a myelitis has been set up, the symptoms will be such as are found described under that caption.

COURSE AND PROGNOSIS. The disease is very slow in onset, and in passing to its successive stages. Patients have been known to survive for twenty years. The prognosis is usually grave, unless it is diagnosed at an early stage. It has been known to stop at any stage, and recoveries are by no means unknown. While pain tends to decrease with the advent of the paralysis, patients have been known to succumb to the distress of the pain and the rigidities.

DIAGNOSIS. The diagnosis of a spinal meningitis of the internal layer of the dura is to be made from the appearance of symptoms of a primary compression of the nerve-roots, especially in the cervical region, without a pre-existing spinal disease to explain the condition. If it is in the later stages, the true origin of the process would be suspected from the fact that the process was a very chronic one, and that the symptoms of nerve-irritation had antedated the paralysis by months or years. When all the membranes are affected, or the cranial dura is also affected, the diagnosis may be impossible. The cerebral symptoms may overshadow the spinal.

DIFFERENTIAL DIAGNOSIS. Syringomyelia. This very closely resembles pachymeningitis cervicalis in its general character, and distribution, since in each case there are muscular atrophy in the arms, and paralysis and wasting in the legs, but in syringomyelia the pains in the arms are usually much slighter than result from pachymeningitis, and there is rarely the early limited impairment of all forms of sense-perception which results from damage to the nerve-roots in pachymeningitis, and syringomyelia is a far more chronic disease.

Diseases of the Spine. These are often capable of exclusion from the history. In addition to this the root-pains are rarely very severe, and tenderness and irregularity of the spinous processes can be discovered by examination soon after the commencement of the disease. This practically differentiates external pachymeningitis also. The X-Ray should furnish data for diagnosis.

Progressive muscular atrophy is free from the severer degrees of pain, and is often without any sensory symptoms, and has none of the stiffness and rigidity so conspicuous in the disease in question.

Myelitis has segmental symptoms before the neuritic ones. It is usually much more acute, and in any case the symptoms of paralysis, and disturbance of the sphincters precede, and do not follow, the pains and changes of sensation. If the invasion has been rapid and widespread, it is impossible to differentiate cervical myelitis from pachymeningitis cervicalis hypertrophica.

Tumor. If it involves the nerve-roots it cannot be diagnosed from a pachymeningitis, since its results are the same, but generally speaking the symptoms of tumor are compression of the cord, rather than irritation of the nerve-roots, and it differs in two other peculiarities: first, that the vertical area of pachymeningitis is considerably greater; and secondly, that the effects are usually bilateral from the first.

TREATMENT. In view of the great probability of a syphilitic causation, lumbar puncture should be the first measure employed. If syphilis does prove to be the cause, the intraspinous use of Salvarsanized serum will be the remedy of election. During any treatment, the posture of a patient with internal pachymeningitis should be such as to drain the spine, and it has been suggested that one of the best methods is to keep the patient as much as possible upon the side or face, and this may be accomplished by arranging a padded board, or a bolster, or other form of bed-rest, against which he can lean. If syphilis is not the cause, warm baths are of value, and hot packs and sweat baths have produced good results. Galvanism to the spine has been employed in such a manner as to include the involved area between the poles. Counter-irritation by Iodine, or the actual cautery, benefits some cases. Operation has been suggested, and should be thought of as a possibility. The danger is, however, far more than that involved in gaining access to the spinal cord, and rests largely upon the probability that the removal of the diseased tissues will cause irreparable injury to the cord.

THERAPEUTICS. Remedies to be used for the condition are the following: Aconite has been advised in the preliminary stages where we get signs of inflammation, and tearing pains in the nape of the neck, with stiffness and pain running to the shoulder, particularly the right. This idea of its adaptability rests upon the similarity of the peripheral pains and numbnesses to those of the drug. These, however, are the results of mechanical compression of the spinal nerves, and only come after the exudation on the meninges has become very pronounced. The congestive stage without exudation, which is the proper field for the use of Aconite, has passed long before the condition is susceptible of diagnosis. Bryonia is always to be thought of in the stage where we find an exudative inflammation of the serous membrane, and the presence of the pain is no bar to the use of the drug. It is probably our most efficient remedy. Cuprum acet. is specifically a spinal cord remedy, with clonic spasms which begin at the extremities, and there is a paralysis of all the muscles of the back up to the neck. There is much nausea, vomiting, and torpidity of the bowels. Ferrum phos. is quoted by Bartlett as a remedy for the early febrile stage, with a soft and full pulse, before exudation has taken place. Hepar sulph. should be thought of when there is suppuration. Oxalic acid when the cord has become affected, and there is a meningo-myelitis as a result. Iodoformum is suggested by O'Connor on account of its favorable action in cerebral cases. Silicea in suppurative conditions generally. Secale cornutum for tenderness of the lower cervical, and upper dorsal vertebræ, with stiffness of the neck, tingling of the back which extends to the fingers and toes, and pressure upon the parts affected causes pain there, and through the chest, which is aggravated by every exertion or strain upon the spine. Agaricus for the cases where the spinal inflammation is combined with that of the cerebral meninges. With the ordinary cerebral symptoms there are drawing pains in the occiput, painful sensitiveness of the scalp, stiffness of the nape of the neck and back, violent pains along the spine, with stiffness and soreness, and liability to convulsions. Cicuta vir. has the same combination of cerebral and spinal symptoms, with a great stiffness of the neck, and tension and soreness of the muscles with a retraction of the head. There are numbness and distortion of the limbs, and a marked concomitant is gastralgia with vomiting.

Physiological remedies are the Bichloride of Mercury, or Mercury by inunction, and the Iodides, largely used in the belief that they are absorbents of new growths and exudates, and also in view of the possible syphilitic origin of many of the cases. The other remedies are sedatives in some form, as no other definite medication has thus far met with acceptance.

Leptomeningitis Spinalis.

This rarely occurs, except as a complication of the form of pachymeningitis just described, or as an extension of the cerebral type, and is sufficiently described under those heads.

CHAPTER V.

INFLAMMATORY DISEASES OF THE SPINAL CORD.

MYELITIS.

DEFINITION. It is a diffuse inflammation of the spinal cord, resulting in the destruction, to a varying degree, of all the spinal elements. It varies greatly in intensity, and also in distribution. Variations in intensity cause it to be spoken of as Acute, Sub-acute, and Chronic, while it is called Transverse when it extends completely, or practically completely across the cord. In this case it is liable to confine itself vertically to one or two segments at the most. It is called Disseminated, or Diffuse when it has a wide vertical distribution, but is limited horizontally, so that no particular segment is extensively damaged, but many are partially destroyed. If it has extended downward from its original focus, it is spoken of as Descending, and a contrary direction gives it the name of Ascending. Locally again it may be Central, Peripheral, or Marginal, Annular, or Unilateral (rare).

THE SYMPTOMS OF A TYPICAL CASE. There is a feverish condition with a very rapid paralysis of the lower limbs, which is spastic in type, with abolition of the deep reflexes, and the incontinence of urine and feces. The muscles of the paralyzed limbs speedily waste, and there is sweating, coldness, and blueness of the affected parts. Bedsores occur upon the sacrum. Sensibility is lost in the paralyzed area, and above the insensitive parts there is a zone of increased sensibility to touch and pain.

DIFFERENTIAL DIAGNOSIS. It is to be differentiated from Spinal Meningitis, Hemorrhage into the Cord, Meningeal Spinal Hemorrhages, Spinal Hemorrhage, Multiple Neuritis, Multiple Sclerosis, Hysterical Paraplegia.

FREQUENCY. It is now considered much more rare than formerly, and this arises from the fact that many diseases, formerly considered myelitis, have been differentiated, and assigned to specific categories, since they have been shown to be selective, and not general, in their destructive action, yet it is one of the commonest of localized organic nervous diseases.

AGE. Early in adult life, twenty-five to forty-five. Children with apparently this condition are generally the victims of meningo-encephalitis, while aged cases actually are victims of a senile form of paraplegia from arterio-sclerosis.

SEX. Largely male, since they are much more subject to the vicissitudes of exertion and exposure, which are such frequent elements in the causation.

ETIOLOGY. The spinal cord is not so susceptible to the invasion of bacteria as is the brain, but the infections and intoxications are usually responsible for the occurrence of myelitis. It occurs after practically all the infections and intoxications, especially after tuberculosis and gonorrhea (Russell-White, and Jelliffe), and I have seen a case occurring in the later stage of measles. Lead, alcohol, arsenic, and coal gas are all possible causes. It may come after parturition, and from furunculosis near the spine (Russell). Traumatism may directly cause it, but it is usually merely contributory. Russell quotes a case where a man, who had suffered some years previously from lead poisoning, within five or six hours after a fall of a few feet developed myelitis. This is also the role probably played by cold and wet as a causative agent. Injuries and exposure both reduce the resistance to invasion. Caisson disease is that form where the cord becomes the site of numerous punctate hemorrhages from the volcanic liberation of the retained gases of the blood, in consequence of a too rapid return to normal pressure, after having been in air compressed to a high degree. Diseases of the spinal bones may extend to the tissues of the cord, and the annular variety of myelitis is an extension of inflammations of the meninges. Sometimes no adequate cause can be discovered.

PATHOLOGY. The essential change is an inflammation, or a necrotic degeneration from vascular obstruction. Softening is the result in either case; there is a fatty degeneration of the myelin, and resulting from this, a swelling and disintegration of the axis cylinders. The gross external appearance of the cord is not altered, but it feels softer than normal, and, on incising the dura, the contents will often well up in a semi-fluid state, and all distinction between white and gray will be found to be abolished.

In old cases sclerosis occurs, and the cord will be shrunken and hard. This general description is of course true only in the acute transverse cases where there is destruction of a complete hemisection, but it is always true of the specific areas affected. No matter how extensive the lesion, some fibres will still persist. Microscopically the vessel-walls are everywhere congested, filled with leucocytes, and surrounded by those which have emigrated, and also round cells, and small nuclei. In many cases bacteria may be discovered. The intensity of the congestion is evinced by the general rupture of the vessels, and small hemorrhages are everywhere present, resulting from the destructive action of bacteria upon the coats of the arteries. Cysts may be a final result. Leucocytes are peculiarly apt to collect about the cells of the anterior horns. Cells are found to be destroyed in all their parts, cell-bodies, dendrites, and axons. From the pressure of the exudates the conduction-tracts are degenerated, and as a result of this pressure the myelin sheath swells, and becomes infiltrated, and the axis-cylinder degenerates in turn. By this cutting off of conduction-tracts, cerebral control is lost, and the local degeneration destroys the efficiency of the segments involved in their rôle of reflex arcs, or secondary or automatic centers.

After this solution of the normal functionating structures of the cord, a sclerosis occurs, which replaces none of them in a proper sense, but substitutes for them an overgrowth of neuroglia-tissue, which is practically the formation of a scar. This is the result of an irritative reaction, which sets up karyokinesis by the multiplication of the neuroglia or Deiter's cells. The bacteria usually found are Streptococcus, White Staphylococcus, Yellow Staphylococcus, and a Diplococcus of a special kind, which has not yet been so accurately differentiated as to be termed the Diplococcus of Myelitis. There are many cases where no bacteria can be found and it would be a question whether the process had been the result of bacterial activity had not Marinesco shown that bacteria may disappear in a few days after they have induced their characteristic pathological changes. If the process is one of softening, while there may be much congestion, the blood-vessels are less distended, fewer white cells are found in the perivascular spaces, the connective-tissue cells are less numerous, the nerve-cells are swollen, and glossy, and stain badly. There is a great deal of granular matter, and nerve detritus, and it is thought to be diagnostic that in inflammatory swelling of the nerve-cells, they will take a carmine stain, whereas in degenerative changes they will not (Dana). It is to be remembered that here, as elsewhere, nerve cells once destroyed are never replaced.

Transverse myelitis in a large way, and other forms as surely, but less generally, cut off fibres from their distant nutrient cells, as well as injure cells at the site of the lesion. After a fibre is thus cut off, a degeneration is set up in the axis-cylinder, which extends in a direction away from the cell-body. Therefore motor degeneration is descending, while sensory is ascending, and the distribution of both these degenerations is greatest at the site of the lesion. We shall therefore find all the motor fibres degenerated below the lesion, and since the association tracts, and the comma tract of Schultze lying in the column of Burdach, and the septo-marginal tract lying in Goll, are also descending, they will also be found to be degenerate. In the lowest part of the cord the sensory fibres are all comprised in the columns of Goll and Burdach, although the tract of Gowers is beginning. The direct cerebellar begins at the first lumbar. Sensory degeneration extends upward, and therefore the higher the site of a lesion in the cord the wider will be the resulting area of degeneration.

Secondary degeneration results from any sort of lesion of the substance of the cord, not only as we have seen in a Myelitis of the primary variety,

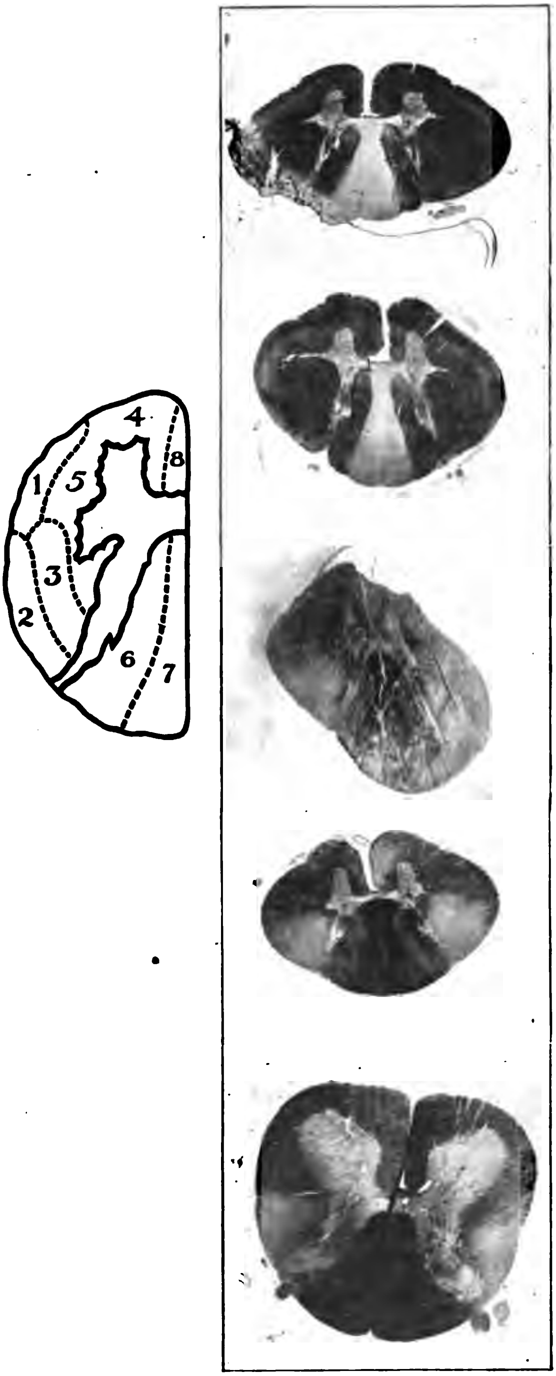


Figure 61. Complete Transverse Myelitis of the Dorsal Region.

Section of the Cervical Cord is at the top, of the Sacral at the Bottom. Referring to the schematic diagram, it will be seen that the destruction of an entire section of the cord produces an upward degeneration of the sensory Columns of Goll (7), Direct Cerebellar (2), and Gowers (1). It produces a downward *motor* degeneration, *i. e.*, Crossed Pyramidal Tract (3). Reference to the schematic half will show the location of the affected tracts.

but also from secondary forms resulting from tumor, caries of the spine, fractures of the vertebræ, hemorrhages either within or without the cord, wounds of the cord. It begins within twenty-four hours of an injury, and is an active process for from six to eight weeks.

SYMPTOMATOLOGY. This is of three classes, paraplegic, segmental, and root, and their relative importance will vary according to the horizontal and vertical extent of the lesion, according to the particular segments involved, and to the degree to which the primary disease has involved the nerve-roots. The gravity of the resulting damage depends largely upon its site, since some levels are far richer in cell areas than are others. Paraplegic symptoms are such paralyses and motor phenomena as are developed by the interference with the conduction-tracts.

Segmental symptoms are such as arise from the destruction of motor, sensory, and trophic cells in the affected segments. Segmental lesions produce flaccid atrophic forms of paralysis in muscles which have been previously furnished with their nerve-supply by the cells of the segment in question. We shall also find sensory disturbances in the areas of skin innervated from these segments.

Reflexes will also be abolished, if any portion of their arcs (spinal cord cells, motor or sensory nerves) are involved in these segmental softenings. It is a point of some diagnostic importance whether the motor or sensory symptoms are accompanied from the first by a paraplegia, or whether that is a later developement. If the paraplegia is an early symptom, we have good reason for believing that the cord is the primary seat of injury, but if the paraplegia comes on later, it must be considered that the injury to the cord is secondary to some other process, and that the cord is only injured by compression.

Root-symptoms are these. Pain is the most constant evident factor, and is usually sharp, neuralgic in character, and sometimes tender points develope. It may be intermittent, or constant, and, if in the limbs, is apt to be referred to the joints. This is at times the only symptom. There is usually an intense hyperemia of the skin, and yet, in spite of this general increase of sensitiveness, we are apt, in the later stages, to find areas of anesthesia to which pain is referred. Such a condition is called *anesthesia dolorosa.*

Spinal pain is due to an irritation of the posterior nerve-roots at their entrance into the cord, or to irritation of the sensory fibres as they pass up in the cord. All the above-mentioned changes of sensation are strictly limited to the areas which derive their fibres from the segments of the cord which are the seat of the disease. Irritation of the motor nerve-roots may also cause painful contractures of the muscles which they innervate, but this symptom is more rare than a gradual loss of power from interruption of the fibres in their course. The interruption of motor fibres causes an increasing degree of muscular weakness accompanied by wasting.

The rapidity of this atrophy varies considerably, and with it the degree of change in the electrical reactions. If the atrophic paralysis is slow, there will be found to be merely a progressive decrease in both faradic and galvanic irritability; if it is more rapid, there is often the reaction of degeneration, and, at times, what is known as the mixed form of reaction, on account of the partial character of the damage of the fibres to the muscles; and a definition of this form is this: the reaction in the nerve is normal, and altered only in the muscles.

The most common form of the disease is a Transverse Myelitis of the dorsal region, and the next is Transverse Lumbar.

Transverse Dorsal Myelitis. This is the most common type and location. In this variety of myelitis, there is a minimum of segmental symptoms, since the segments at this level supply only the muscles of the abdomen, and the lower thorax, and losses must be very complete to be conspicuous. We are mainly impressed by the following: loss of voluntary motion in the lower limbs; loss of sensation in all parts below the level of the lesion (usually accompanied by a zone of hyperesthesia, about two inches in width, just above it); loss of control over micturition and defecation. There will be a variation in the degree of completeness of such losses proportioned to the completeness of the lesion. When the lesion is incomplete, there may be a disproportion between losses of one variety and those of another, but it may be broadly affirmed, that with a variable degree of injury to the tracts of the cord, the motor palsy will be greater than the loss of sensation, or of control over the functions of the bowels and bladder. At times the sensory change is an erroneous localization of sensory stimuli.

The paralysis of the lower limbs (paraplegia) is at first a simple loss of power, which developes into a paralysis of the spastic type, i. e., the limbs grow more and more rigid, and draw up spontaneously, or when manipulated, and eventually become more or less completely fixed in some degree of flexion. This is due to two causes: the first is a loss of inhibition over the unrestrained reaction of the motor cells of the anterior horn in answer to the peripheral stimuli, which are always inciting them to reflex action, and which in health is regulated by the corrective influences sent down in the motor tracts, which by this disease have been cut off; secondly, from the fact that from the lack of nutritive stimuli, exerted through these same motor tracts, the motor cells are in a state of anemic irritability. Knee-jerks are exaggerated, there are ankle-clonus, and the Babinski toe-sign. For reasons which are still under discussion, a complete division of the motor tracts by myelitis produces a picture differing from that which we might expect. In such cases the knee-jerks are not exaggerated, but are abolished; perhaps because muscular tonus, which in the incomplete lesion was still in some measure distributed to the motor cells of the anterior horns, is now completely cut off. The converse of this, viz., that, if the knee-jerks are absent, we have proof that the existing myelitis is complete, is not always a truth. In the early stages of a myelitis, such a total loss of knee-jerk may be the temporary result of the shock of the invasion, or as Russell points out, in cases where the onset is rapid, the paralysis may be flaccid for the first few days, instead of spastic, as would be expected. This is due to the fact that in this preliminary stage the pyramidal tracts are irritated, thus temporarily increasing the inhibitory power which is soon to be lost. The muscles retain their volume, and normal electrical excitability, even after paralysis has existed for a long time. There is anesthesia, and girdle-pains about the waist may be present, and, if the posterior nerve-roots are especially attacked, there will be severe lancinating pains in the area of the distribution of these nerves (root-pains).

Retention of urine is usually an early symptom in severe cases, and must be relieved by the use of the catheter, but, as the case goes on, this condition may be replaced by an incontinence, due to paralysis of the sphincter. Priapism is often present, it is common in cervical, frequent in dorsal, but absent in lumbar localization, and impotence is also found in some cases.

The bowel shows the same aberration from the normal, in that a primary retention of the feces is likely to be followed by involuntary and unconscious discharges. In lesions above the mid-dorsal region we find much gaseous distention of the bowels, and the bladder has been known to rupture. Bedsores are common, and develope with great rapidity, beginning with a patch of erythema, which assumes a darker color in the middle, followed by blebs filled with fluid, which rapidly break down, and reveal a deep-seated and extensive slough, which often reaches down to the bone. They are caused by the disorganization of the trophic elements in the cord, but they may be avoided, in some cases, by surgical cleanliness, and certainly are favored by the irritation and moisture resulting from imperfect care of the excretions. Vasomotor and secretory disturbances are present; edema, arthropathies, anidrosis, etc.

Lumbar Myelitis affects segments which are motor, sensory, and trophic to the lower limbs, and leave the power and sensation of the trunk unaffected, or, at the most, impairing it at its lowest levels. There will be paralysis of the lower limbs, which will be flaccid in type, that is, the legs will hang powerless, with the knee-jerks abolished, as well as all other deep reflexes; the limbs feel hot for a few days, but after this they are cold. The skin reflexes are also lost, and since the nourishment of the tissues depends in some way upon the integrity of the anterior portions of the cord, there will be wasting, and the skin will become rough and congested, and there is generally a cold sweat. There may be a lack of completeness in the lesion, and if the upper part of the lumbar portion of the cord is healthy, the knee-jerk will be spared, or even exaggerated, as a phenomenon of irritation, since the ilio-inguinal, the crural, and the obturator nerves are not involved in the area of destruction. There will be anesthesia of the legs, and perhaps a slight involvement of the lower abdomen. There will be no girdle-pains in this case, but instead, there will be radiating pains in the distribution of the nerves of the extremities. The paralysis of the bladder and rectum will be very pronounced. There is a group of cases where the paralysis gradually appears, first in one, and then in the other limb, and finally developes into a typical paraplegia. These cases are called Chronic Transverse Myelitis, but will probably develope into a Multiple Sclerosis.

Sacral Myelitis produces these same anesthesias, overflow, incontinence of the bladder, and no power in the rectal sphincter. The over-distention of the bladder favors the developement of cystitis. Being below the origin of the nerves to the lower extremities, there is no paralysis of the lower limbs, if the myelitis is strictly limited to these segments.

Myelitis of the conus terminale gives rise to many of the symptoms which we should naturally ascribe to a lesion of the sacral portion of the cord, since it is composed of the mass of nerves from these segments, just before they are spread out in the cauda equina. There is paralysis of the bladder and rectum, the patient is impotent, there is anesthesia of the anal, scrotal, and perineal regions, of the penis, of the upper and inner surface of the thighs, and sometimes a degenerative paralysis of the sciatic region.

Cervical Myelitis. The symptoms here will differ, according to the involvement or escape of the cervical enlargement. When this is involved, the arms will naturally show an atrophic, flaccid paralysis, while the legs will be spastic, since the cutting off of the motor tracts anywhere above a reflex center takes away inhibitory control, but a segmental loss results in atrophic paralysis. There will be anesthesia of both arms, and of all parts below the lesion, as in all other localizations, but not always complete, since some fibres may remain intact in almost every localized inflammation. This lack of sense-perception comprises all varieties, tactile, pain, and temperature. Respiration may be imperfect, from paralysis of the diaphragm through the phrenic nerve. There may be pupillary symptoms, since the movements of the pupil are strongly influenced by the condition of the lowest cervical sympathetic ganglion, which gains its fibres from this, and the upper dorsal segment. The pupil is usually dilated, and in disseminated cases optic atrophy is common. Dysphagia, hiccough, variation in pulse (slow, or fast, or irregular), and hyperpyrexia.

If the lesion is above the cervical enlargement, the direct motor and trophic innervation of the arms and the intercostals escapes; that is: it is no longer a segmental loss, and so there is no atrophy, but the arms are the seat of a spastic paralysis, while respiration is still affected by the paralysis of the diaphragm through the roots of origin of the pneumogastric, and, if the lesion is still higher, the medulla is involved.

Incomplete Transverse Myelitis is not uncommon, and then the picture is confusing from the incompleteness of some, and the absence of other symptoms. Paralyses may not be complete; rather those degrees of motor loss which are termed "Pareses." Instead of anesthesia, we find hyperesthesia, which is a diminution, instead of an actual loss of sense-perception. In other cases, the sensory loss will be confined to one side of the body, and sometimes the lesion is so strictly confined to one side of the body that we find a perfect picture of what is termed "Brown-Sequard Paralysis," which is this: on the side of the lesion is a motor palsy, while the anesthesia is on the other, yet with this reservation, that muscle-sense is lost on the paralyzed, and preserved on the opposite side. This sensory phenomenon is supposed to owe its existence to the fact that all sense-perceptions, except those from muscles, and tendons, and joint surfaces, are conveyed in tracts, which cross to the opposite side of the cord, immediately after their entrance. Mott and Turner (Oppenheim) have not been able to verify this histologically, but it seems to have been clinically established. On the paralyzed side, there seems to be some sensory exaltation, especially to pain. Temperature is generally raised from ½ to 1°. The functions of the bladder and rectum are usually, but not always, disturbed. On the paralyzed side, after a little interval of loss, the knee-jerks are exalted. On the paralyzed side there is a half-girdle of hyperesthesia, and pain, while on the other side there is a half-girdle of anesthesia with a border of hyperesthesia above it.

It is a rule to be remembered, that all signs of a transverse break in conduction are present, if part of them only are suggested. The vertical extent of the process may be very considerable, e. g., the whole dorsal, or parts of the dorsal and lumbar may be involved, and, of course, such an extent of lesion may modify the symptomatology.

Disseminated Myelitis. This is a form of myelitis where patches and tracts of inflammation are scattered throughout the cord, and even involve the medulla, and the brain. It is evident that the lesions may be so distributed, that unitedly they might constitute a complete cross-section, and the symptoms, so far as they relate to conduction, will be similar to the transverse form, but diagnostically they will show very few completely marked segmental symptoms. The causation is an infection or intoxication, from whose general diffusion small foci of inflammation are set up at numerous points.

Many cases are atypical, and often symptoms flowing from implication of the brain, or medulla, will precede those from the spinal cord. There may be initially the paralysis of an arm, or of a cranial nerve, or the onset may be like an apoplexy. The involvement of the brain may cause symptoms like those of chorea, but, as in other forms of myelitis, a paraplegia is apt to be the prominent symptom. Optic neuritis has been known to occur. We find intention tremor and ataxia in both, and it is said that some cases develope into a multiple sclerosis, but such a statement is a confusion of terms. The temperature is generally normal, but in some cases there is a systemic reaction to the infection, and for a variable time it may show an elevation of from 4-6°.

Compression Myelitis. DEFINITION. Certain cases of chronic myelitis arise from no disease of the cord itself, but are the results of compression by some conditions arising in the structures outside of it.

ETIOLOGY. A very common cause is caries of the vertebræ, or tuberculous disease of them, or traumatism which dislocates or crushes them. The usual result of disease of the bones is an extradural purulent exudate, which encroaches upon the spinal cavity, and compresses the cord. Without this purulent result the bones may break down, and assume new positions, which encroach upon the cavity of the column. In case of traumatism there may be a subluxation of the bones, or parts may be driven into the canal (laceration of the cord is excepted from this discussion), or the inter-vertebral cartilage may be displaced inward, or the resulting hemorrhage from the accident may form an encroaching mass. Tumors may arise from the bones, or from the periosteum, or from the membranes. Aneurism of the descending aorta may erode the bones of the lower half of the dorsal region, and allow them to sink inward, and a cancerous growth of the posterior mediastinum has infiltrated the tissues, and invaded the cavity in a case seen by the author. Echinococcus cysts have also been the agent for the production of pressure. All these conditions will not infallibly produce a myelitis, but are sufficient causes if the pressure is sufficiently severe and of long duration. The spine may be injured or seriously diseased without a compression myelitis being the necessary result.

SYMPTOMATOLOGY. This must conform to those just given, to enable us to state that a myelitis has resulted. Root-pains are common in both conditions, and if they occur before the paraplegia, we must consider that the causative condition is without the cord.

COURSE, PROGNOSIS, DIAGNOSIS AND TREATMENT OF ALL FORMS OF MYELITIS.

COURSE. The onset may be very acute, needing the passage of only a few hours to develope the complete symptom complex. Such a case is sometimes termed Myelomalacia, or Spinal Softening. Usually from one to ten days are consumed in the process, and chills and fever show that there is an inflammatory process from some source of infection. Starr quotes a case of disseminated myelitis which took twelve days to develope the paraplegia, and one of the transverse type in the lumbar region where it was complete within four hours of the onset. With, or before the chill, there is a feeling of numbness in the legs, followed by an exaggeration of sensibility, which is later replaced by an abolition, or reduction of it. The invasion period is sometimes marked by pain, but it is never a pronounced feature. There is next a weakness in the lower limbs, which speedily becomes a paralysis, but some cases are very insidious, and in non-fatal cases the developement of the disease is very slow. In some cases there may be only a weakness of the legs, always with some stiffness, and the sensory changes are equally slight. However slow the onset, disseminated myelitis will finally display to some extent the more definite symptoms of a localized myelitis.

PROGNOSIS. Typical cases are usually fatal, but a difference in etiology produces a difference in symptoms, and degree of severity, and probability of fatality. Some result in a myelitis which tends to recovery, while others result in chronic forms, and still others to a type marked by remissions and relapses. The results of treatment in cases of gradual compression have been very gratifying, but in traumatic cases relief of the pressure must be within three hours of the accident, or no hope can be entertained of restoration of function. Even then the prognosis is very bad, on account of the probable laceration of the cord. In any case, marks of the damage will be persistent, although function may be very completely restored. Extensive motor paralysis has a worse prognosis than an equal sensory loss, better if the lesion is in the dorsal region than when it is in the cervical. Generally the infectious cases, such as result from variola, typhoid, erysipelas, influenza, etc., and which resemble an acute ataxia, tend to recover. Gonorrheal cases almost universally do well, as do those arising from malarial poisoning, but these latter are marked by a strong tendency to remittency. Syphilitic meningo-myelitis is not necessarily fatal in its outcome. Some cases complicated by meningitis, and neuritis, have recovered. Acute, or incomplete cases, are more favorable in prognosis than those of insidious onset, or the completely transverse. The slender, and the aged, bear the disease better than the robust, and the youthful. The return of sensation to an anesthetic part is a sign of recovery, while an extension of this loss is of bad omen. Bed-sores, and complete paralysis of the bladder and rectum are bad signs. The longer continued the case, the worse the prognosis, and death usually results from bed-sores, or cystitis. An attack coming on in the puerperium, or in the course of tuberculosis has a bad prognosis. However great the degree of recovery, some amount of spastic paraplegia will remain in the lower limbs.

DIAGNOSIS. This rests upon the fact that suddenly, or in a few days, a person developes a paraplegia, with exaggeration of the deep reflexes, the only exception being, when the segments containing the reflex arc are destroyed, and then in that particular reflex only, we find complete loss. This is a segmental symptom, and to this are added root-symptoms, if the cause of the myelitis originates in the meninges. The functions of the rectum and the bladder are disturbed; there is atrophy of the lower limbs, and blueness and coldness of the surface, and profuse sweat. Bedsores almost always occur. Whether the lesion in the cord be complete or incomplete, there is a persisting plantar reflex, of the extensor type.

DIAGNOSIS OF SITE. The upper level of the anesthesia affords the best means of estimating which segment is the uppermost one affected. This border is apt to be definite in outline, but if above it, as is common, we find a zone of hyperesthesia an inch or an inch and a half in width, we may be sure the lesion is not higher than the segment just below the one which gives off the fibres to the hyperesthetic zone, since this is a symptom of irritation, and not of destruction. It is not so easy to locate the damage by the impairment of muscles, since each muscle, though mainly innervated from a definite segment, is partially innervated by others in the neighborhood. In case the lesion is between the seventh and twelfth segments, Beevor's Sign may be found; i. e., if the patient is told to fold the arms, and rise from a recumbent to a sitting posture, the umbilicus will be found to ride upward to the diaphragm, owing to the fact that the unparalyzed abdominal muscles are unopposed. Determination of the lowest border of the injury is less definite, and we can only say with certainty, that if the bladder and rectum are still tonic, the lesion is above the lumbar enlargement.

DIFFERENTIAL DIAGNOSIS. Spinal Meningitis is usually secondary to meningitis of some other part of the cerebro-spinal axis. When the cerebrum is primarily the seat of the disorder, the spine may not attract notice, but if the spinal meninges are the primary location, we shall find more pain, rigidity, convulsions, and fever, than are usually found in a myelitis.

Hemorrhage into the Cord is, in its inception, very different from myelitis. The patient gets a paraplegia, but not in a few days or hours: on the contrary, he is apt to drop as if struck, and a definite spot in the spine is the seat of an acute pain. In myelitis, pain is not a pronounced symptom, and, if present, is not necessarily confined to the spine. After a few days the symptoms are similar, since the hemorrhage sets up acutely the same necrosis in the substance of the cord which occurs more slowly in a typical myelitis.

Spinal Meningeal, and Spinal Hemorrhage, differ at their onset from myelitis in possessing the same instantaneousness of attack that has been spoken of under the preceding heading, but more exactly speaking, rapidity of onset differentiates hemorrhage into the cord, and myelitis. In spinal meningeal hemorrhage there is likely to be a pain in the back in a definite spot, and root-pains are noticeable, since primarily from the meningeal site, or from a secondary implication of the meninges, the roots are involved. From this factor we get more initial rigidity of the muscles of the back than in myelitis. The final result of meningeal hemorrhage is the production of a compression myelitis. Lumbar puncture revealing blood diluted with serum indicates spinal hemorrhage,

Multiple Neuritis. This typically involves all four extremities, and if the face is affected, it is surely not a myelitis. It is a peripheral disease, and if it involves the lower limbs alone, it is distinctive, in that it leaves the muscles near the trunk free, or it is true up to a period of very complete developement; in myelitis the whole musculature of the limbs affected is paralyzed together. The bladder and the rectum are practically never involved in multiple neuritis. The muscles are tender in neuritis, and not in myelitis.

Multiple Sclerosis. Owing to the similarity in the site and distribution of the lesions, it is not surprising that there should be points of similarity in the symptoms, although multiple sclerosis is degenerative, while myelitis is inflammatory. The onset of myelitis is abrupt, and may be marked by chills and fever, while that of multiple sclerosis is gradual, without such accompaniments. This difference of onset, and the rate of subsequent developement, may be the only distinguishing mark in atypical cases. Both have an ataxia, and paraplegia, and both a tremor, but it is very rare that it is of the intention variety in myelitis. Myelitis never has the staccato speech, nor the nystagmus which are symptoms in typical cases of multiple sclerosis.

Hysterical Paraplegia. The patient is generally female, and youthful, and an emotional shock can be discovered. The paralysis is almost always a monoplegia, instead of the customary paraplegia of myelitis; the knee-jerks are only slightly exaggerated, and Babinski's reflex is never present. There are rigidities instead of spasticities, and the mental origin of them can be demonstrated if the paralyzed limb is raised from the bed. If the sustaining hand is unobtrusively removed, the limb will not drop on to the bed, but will sink down in a hitching manner. There will be no electrical changes, and the sensory changes will be anesthesias of whole limbs, or considerable portions of a limb, or rectangular patches irregularly disposed over the body, without reference to nervous distribution. Specific atrophy is rare, bedsores are absent, and there may, or may not be incontinence.

Acute Non-progressive Disseminated Encephalo-myelitis. This is a poorly defined form of disseminated myelitis, which is not so fatal as the acute form of disseminated myelitis, and has been termed by von Leyden, "Acute Central Ataxia." It is apt to occur in the course of, or soon after the acute infections, smallpox, typhoid, and variola, and so is most often seen in children and young persons. On account of the severity of the initial disease, the early symptoms are apt to be considered to be unusual symptoms of the primary disease, but, as it passes away, the patient is found to present the following symptoms: loss of speech; loss of power in the limbs; changes in tendon reflexes. Ataxia may be the first symptom, or it may follow unconsciousness, restlessness, or delusions. In the ataxic state there may be intention tremors of the limbs and head, disorders of speech, especially of the scanning variety, but there may be dysarthria. Nystagmus appears to be uncommon. Aphasia, or optic neuritis, sometimes occur in the earliest stages. Death is rare; a few cases recover quickly, and in others some symptoms, especially speech disturbance, persist. There are many points indicating that it should develope into multiple sclerosis, but German authorities deny that this does occur.

TREATMENT. The treatment of this condition is not to be approached with a great deal of optimism, since it is not very efficacious as a rule. Most drugs have been found to be useless.

From the very first, absolute rest in bed, even when voiding secretions. Cupping has been recommended for the early stages, when marked by pain, or the same effect has been obtained by ice-bags, hot fomentations, hot sandbags, or salt-bags. The application of heat may be more pleasant if bran-bags are used, but they must be frequently changed. The wide diffusion of the electric current makes it possible to make use of the various kinds of electrical substitutes for these before-mentioned appliances, and they have the great advantage of being light, and manageable, and the greater merit of needing no renewal. If the case follows an infection or an intoxication, free sweating should be maintained, to aid in eliminating the poison. Vesication has also been used, but it should not be over anesthetic zones. Iodine is a good revulsive in these cases. We may obtain the two-fold advantage of free diaphoresis, and local heat, by the use of the wet pack in the early stages. Hot baths should be avoided, and warm baths only after the disease has ceased to progress (Oppenheim). ("Either cold, 60°, or hot, 100°, are bad." Starr.) Experience has shown that cold applications are more generally valuable and grateful than hot ones, except in those cases which have arisen from exposure to cold. The bowels should be moved every other day, and the catheter should be used with extreme attention to asepsis, and the scrotum should be carefully protected with cotton, to avoid the retention of dampness in its folds. The possibility of bedsores should be ever before the attendants, and their appearance should be combatted by cleanliness, to a surgical degree, careful drying of the parts from body-sweats and the secretions, dusting with zinc, or other powder, and an avoidance of wrinkles in the bed or body-clothing. Greatest in value of these is the frequent change of position, and most efficient of all is the use of a water-bed. When the skin becomes dark, and a slough seems imminent, bathing with cologne, or alcohol, weak solutions of tannin, or alum, temporary applications of dry heat, or positive galvanism may abort it. Painting with weak nitrate of silver solution may help it. If it is possible the continuous bath may be employed. If a bedsore actually forms, it should be dressed with zinc oxide ointment, or a dusting powder of aristol if it remains clean. A septic sloughing sore should be swabbed out thoroughly with hydrogen peroxide, and if that is not efficacious, with bromine. In more chronic stages a sojourn at some warm springs may be of advantage, but water at a high temperature, and sea-bathing should be avoided. If cystitis appears, the bladder should be washed out once or twice daily with a solution of Permanganate of Potassium in a 1-8th to 4% solution, or Boric acid 10%, at 98°, two or three times a day, Salol grs. 5, or Urotropin 10 grs. every four hours by mouth. Other drugs may of course be applicable. The most perfect action of the bowels, the kidneys, and the skin, is essential, not only for general reasons, but also because their perfect function seems to actually drain the spine.

The reflex jerking of the legs is a common and distressing symptom, which may prevent sleep. Hot baths, hot applications, and very mild massage will sometimes help this condition, but by no means certainly, as it is an organic reflex. Cuprum and Zinc should be thought of here. Bromides and Hyoscine hydrobromate are the physiological remedies. Spontaneous contractures may be unavoidable, but they should be combatted by gentle massage and electricity (neither, in the early stages of the disease), and passive movements should also be used. On the other hand we should just as sedulously guard against drop-foot. My usual plan is to support the bed clothes on a cage, or place the limb in an easily removable splint. As strength returns in the limbs, the patient should endeavor by concentration of mind to produce motions with the affected limbs. Atrophied muscles may be energized by injections of nitrate of Strychnia, deep in the muscle.

Electrical Treatment. During the acute stage of the disease electricity has no place, but is valuable in the treatment of the resulting paralyses, anesthesias, or contractures. While it has been advised that the cathode of the galvanic current should be placed over the segments involved in the disease, and the anode upon the abdomen, it is a rather better plan to place the anode upon any part paralyzed, and so complete the circuit. In using this current no interruption should be made until all signs of inflammation have disappeared, and the current at the beginning should be just sufficient to redden the skin at the cathode, and should not be passed for more than five, or at the outside, ten minutes at a sitting. When the condition has become chronic, and the patient has borne this moderate current without destruction of tissues or aggravation of symptoms, the current should be increased up to the point of vigorous contraction of the muscles involved, and for that purpose the polarity should be reversed, having the cathode over the motor-points. The sphincters of the rectum and bladder are incompetent to some degree in all cases of myelitis, and should be the subject of treatment, as soon as it is instituted for any paralytic loss. This is best treated by placing the anode upon the back just at the lower border of the shoulder blades, and the small cathode should be placed upon the anus, and the interrupted current should be passed between the two. The urethra may be similarly treated by introducing a steel cathode into the urethra, and passing a mild current between the two, but care must be exercised that no electrolysis is produced. Paralysis of the bowels may be favorably affected by putting the negative electrode upon the anus, and the positive swept about the bowels in the direction of the large intestine. The effect is sometimes greatly increased by giving a high enema of normal salt solution, and introducing a hollow anodal electrode into the rectum, and using the cathode upon the abdomen. After the muscles have developed an easy response to the galvanic stimulation, faradism may be substituted with probably equally good results, and is often more feasible in cases where patients are not possessed of ample means. The faradic is also the best for treating the areas of anesthesia, where it is applied by means of the one pole being placed upon any indifferent point, and the other being applied to any anesthetic spots by means of a wire-brush electrode. The abdomen may be treated with the static spray in cases of intestinal paralysis, and the same treatment has been applied to the perineum for incontinence. The high-frequency effluve, administered with a glass rectal electrode, has been quite successful in my hands in the treatment of fecal incontinence. The contractures have been benefitted by continuous galvanic currents to the spine, including the diseased segments between the poles, terminating the seance with short, sharp interruptions, and then applications to the muscles.

THERAPEUTICS. It must be recognized that the condition is at first an acute inflammation of nervous tissue, and that is the point in the disease in which our therapeutic measures are most likely to be of advantage. In addressing ourselves to this condition, experience has taught that the most success attends our efforts when they are directed along causal indications. If the cause has been an injury, we should give Arnica, Aconite, or Bellis perennis, Hypericum, and sometimes Belladonna. If it follows closely upon exposure to cold or wet, Dulcamara or Rhus tox. While we are giving these remedies we should not neglect to remove any acute inflammation, or source of infection, to which the myelitis may be secondary. At times it is difficult to form any opinion of the etiology, and, in such case, Mercurius in the 3x has been reported to have been very-efficacious. It certainly exhibits in its symptomatology a progressive paralysis of the extremities, of the bladder, and rectum, with pains in the spine which are aggravated by pressure, and anesthesia in the limbs, and there is a tendency to painful spasms of the muscles. If there is a possible syphilitic causation, a mixed treatment should be energetically inaugurated, or Syphilinum, in a high potency, given. If the invasion has the rare localization of the cervical portion of the cord it will be characterized by a violent priapism, and in this case Picric acid is the best remedy which we have for the condition. Remedies of a more general character are Cuprum arsenicosum, which has the symptoms of myelitis, with the spasmodic action of the limbs very strongly marked; Arsenicum, which is to be thought of for organic cases where the cord symptoms alone are prominent. Oxalic acid is to be used in its place where the meninges are also implicated; this will be shown by the presence of root-pains, and tenderness in the back; this remedy, however, seems to have a very wide application to all degenerative and inflammatory conditions in the spine. Nux vomica is also a remedy indicated where there are paralysis, sensory changes in the limbs, and root and girdle-pains, showing that the meninges in this case also are affected. The applicability of Rhus to organic conditions is very doubtful, since the pains which have been described as root-pains in its pathogenesis, seem rather more probably to be located in the muscles than in the nervous structures. Artemesia abrotanum has aching in the back, with numbness and paralysis of the limbs, and has some clinical evidences in its favor. Phosphide of Zinc should be thought of where the spinal pain is excessive. In organic conditions which seem to be in a condition of stasis, Nux vomica, Ignatia, and Strychnia in all potencies, and in the dosage seeming to be applicable to the given case, are the best remedies. Strychnia phos. 3x has often been of advantage. Other remedies to be considered are Bryonia, Cicuta, Agaricus, Gelsemium, Phosphorus, Physostigma.

Physiological Remedies are mainly the use of Bromides for the cramps in the muscles, and sometimes Morphine and Hyoscine are used for the same purpose, but the Bromides are the better of the three. For cases arising from infectious causes, use is made of the Salicylates, and mixed treatment if it is the result of syphilis, or better Salvarsan, or Neo-salvarsan. Mercury has been used for cases of all descriptions, on account of its action upon interstitial nervous tissues, but its value is considered to be very problematical. Quinine and Arsenic are advised for cases arising from malaria. Ergot has been used extensively for all spinal conditions, but has not proved to be of as much value in myelitis as in some other diseases. For the resulting paralysis Strychnia is almost universally used, either hypodermically, or by the mouth.

ANTERIOR POLIOMYELITIS ACUTA.

Infantile Spinal Paralysis—Acute Atrophic Paralysis—Heine-Medin Disease.

Definition. It is an acute, infectious disease, caused by a definite micro-organism, general in its incidence, especially affecting the central nervous system, but also implicating the parenchymatous organs, and the lymphoid tissue. This is either the effect of the direct action of the virus, or is caused by a secondary toxic substance produced in the cord. It may be either sporadic, or of epidemic distribution, giving rise to an immediate, but temporary, flaccid or mixed motor paralysis, soon to be replaced by a selective and permanent paralysis of relatively few muscles, which undergo a rapid atrophy. Persistent sensory symptoms are very rare, but for the first few days there may be pains in the back and the affected muscles, which latter may remain tender to touch for a considerably longer period. It is more than a spinal paralysis, for the bulb alone may be attacked, and cases have been seen where a facial paralysis has been the only sign of the disease (Cassirer).

Symptoms of a Typical Case. A child of two or three years of age, enjoying previous good health, and of robust constitution, becomes peevish, restless, and irritable. It complains of pain, and the limbs will be found to be tender to touch, and the child soon developes a fever, sometimes accompanied by vomiting, and less often by diarrhœa, but sweating is a very constant symptom. For two or three days the child continues in this condition, when the fever disappears, and the child, who was irritable or torpid, becomes mentally normal, and physically quiet, but is found to have a flaccid paralysis of one leg, perhaps of both, less often in the arms, but possibly hemiplegic, or quite general in distribution. After a week or two, the general paralysis has been replaced by the paralysis of a few muscles, or at the most of one limb, and the parts now paralyzed will rapidly become wasted, cold and blue, and will not develope equally with the normal limb, and may be subject to contracture.

DIFFERENTIAL DIAGNOSIS. To be diagnosed from Palsies of Cerebral origin, Transverse Myelitis, Pseudo-hypertrophic Paralysis, Progressive Muscular Atrophy, Hemorrhage into the Anterior Horn, Cerebro-spinal Meningitis, Multiple Neuritis, Rheumatism, Rachitis, Congenital Dislocation of the Hip, Hip Disease, Erb's Palsy.

AGE. The most susceptible age is the latter half of the second year of life, and 90% of all cases are under five years of age. 95% are in the first decade of life, but no age is immune, as Collins reports a case at the fourth month of life, another case at the 12th day is on record, and Fraenkel and Onuf cite a case which was either intrauterine poliomyelitis, or a central neuritis. Congenital club-foot has been ascribed to antenatal attacks, and a deficient developement of the sacral cord has been discovered in such cases. On the other hand, it has been seen in adults, especially in epidemics. One of the author's cases was a female of thirty-five years of age, and Wickman reports a case of forty-six years. In fact no age is actually immune.

ETIOLOGY. It has been considered that epidemics most commonly occur in heated weather, following cold and wet periods. This amounts to saying that it occurs mostly in summer and fall. It was also held as a fact that cold weather inhibited the contagion. This has not always proved true. It also seems independent of surroundings, and it occurs in open country fully as often as in cities, in children in easy circumstances rather than poverty, in those in previous good health rather than delicacy. Sanitary precautions do not seem to be a very efficient guard, and unsanitary conditions are by no means a demonstrable cause of its prevalence, and broadly speaking, neither city nor country grant immunity. As an instance of the inadequacy of weather or locality as a determining factor, it may be recalled that the epidemic in New York in 1906 occurred in a summer which was conspicuously dry and cool; there were many cases in the summer of 1910, in the Adirondacks, during a season which was particularly salubrious. Its etiology baffled inquiry, until Flexner discovered a specific germ, and the method of infection. He says (American Journal of Diseases of Children, May 9th, 1915), substantially as follows: The virus, or microbic agent consists of minute globules, capable of being viewed under the high powers of a microscope. It is carried by persons, not insects. The virus is not injured by a solution of 5-10% carbolic acid, freezing for 40 days at a temperature of from 2 to 4° C. does not materially affect it, it resists heat more feebly, and can be destroyed in a half hour by subjecting it to a temperature of from 40-50° C. Hydrogen peroxide in a 2% solution kills it, and so do solutions of menthol, and corrosive sublimate. When enclosed in an albuminous envelope, it will withstand drying for several weeks, and therefore saliva or nasal mucus is capable of conveying the infection both in the dry and moist state, and hence it may gain the upper respiratory tract of the victim by the spray produced by the cough, sneeze, or loud conversation of the carrier or invalid, and also, but less certainly, by the dust of the infected place. Small-pox and measles are the only infections which can flourish in every soil, all others requiring favoring accessory conditions, and so in this instance animals and fowls, excepting monkeys only, do not seem to be susceptible to this virus. Aside from monkeys therefore, animals are only passive carriers, if at all. It is extremely persistent in the nasal secretions after an attack, it having been found in the nasal secretions of a monkey five months after recovery. It does not seem to be so persistent in the human subject. It must therefore be concluded that it is usually communicated by human beings in such a manner as to infect the upper air-passages of other individuals, among whom a small portion, being susceptible to the virus, acquire the infection, and develope the disease. The clinical form or variety which may result may be frankly paralytic, the meningitic, or the abortive or ambulatory, in which form no severe symptoms whatever appear. In whatever form, however, the persons may be affected, they become the potential agents for the dissemination of the virus of poliomyelitis, as do some among the healthy persons who have been in intimate contact with those who have been ill. The proof of this lies in the fact that if the blood-serum of one of any of these classes of persons is mixed with the filtered virus of poliomyelitis and the mixture is incubated at 37° C. for a few hours, the serum will neutralize the virus, which is not true of the serum of persons who have not had poliomyelitis. These several classes of infected, or contaminated persons constitute the active agents by whom the virus is spread, and to the control of whom the sanitary measures designed to prevent epidemics must be directed.

Mode of Infection. The upper respiratory tract is the site of both the ingress and the egress of the virus. The lymphatics of the nasal mucosa are in almost direct connection with the subarachnoid space. This was demonstrated by Flexner with the diplococcus intracellularis, when monkeys inoculated intraspinously exhibited the diplococcus subsequently, both in leucocytes, and free in the nasopharynx. The virus of poliomyelitis has been demonstrated in the nasopharynx of infected monkeys. Successful inoculation of monkeys with the virus of poliomyelitis has been done via the pharyngeal mucosa. Numerous investigators have also shown that the tonsils of human beings may contain the virus of poliomyelitis.

Immunity. Experimentally, Flexner and Clark have shown that one attack confers comparative immunity, through the formation of antibodies, and clinically it has been established that a second attack is extremely rare. Apparent relapses do occur in some cases in the few following weeks, generally taking the form of a wider extension of the paralysis. Gowers reported this as occurring in only 1 out of 116 cases. The most direct route to the nervous tissues is by the mucosa of the nasopharynx, because there is direct communication between it and the meninges by way of the lymphatics, which pass with the filaments of the olfactory nerve through the cribriform plate of the ethmoid bone. It has also been found that this is the path of the elimination of the virus into the external world, since it is a necessity for further infection that the virus must be extruded in a viable and transmissable condition. The tracheal mucosa, and that of the lungs, do not afford a point of easy entrance unless gross lesions previously exist, and large quantities of virus can be introduced into the stomach, or duodenum without the production of a spinal paralysis, if only peristalsis be preserved. If peristalsis is paralyzed, infection will readily take place. This explains why previous gastro-intestinal disease predisposes a person to infection by the germs of anterior poliomyelitis.

PATHOLOGY. Owing to the fact that few patients die in the acute stages of the disease, the pathological findings have generally been confined to the discovery of atrophic scars in the anterior horns of the cord. This led to the view, which formerly was universal, that the paralysis was the result of an embolic plugging of one or more of the anterior spinal arteries, resulting in the necrosis of nervous tissues, and the later developement of interstitial scar-tissue. It is now definitely established, largely by animal experimentation, since the discovery of the specific virus, that the disease is essentially a general infection, with definite and constant changes, not only in the cord, not only in the central nervous system as a whole, not excepting the peripheral nerves, but in the viscera also. The virus, obtaining access to the cerebrospinal fluid by way of the nasopharyngeal mucosa, localizes first upon the leptomeninges. A reaction quickly ensues, which is shown by the changes in the cerebrospinal fluid. In 24, 48 or 72 hours a lymphocytosis occurs which produces a slight opalescence of the fluid, which has now become subject to spontaneous coagulation. This condition of increased protein content is of brief duration, and is followed by a stage where the fluid is clear, and limpid, and noncoagulable, but with an increase of lymphocytes. Lymphocytosis occurs here more quickly than in the blood. In monkeys this is the state of the fluid coincident with, or appearing soon after, the onset of the paralysis. Taken from a monkey, the fluid, *if clear and limpid,* will not convey the infection, or produce paralysis, and while it may be in excess, and may contain an increased number of lymphocytes, it is non-infectious even if injected into the brain. As a result of this infection we also find a hyperemia of the leptomeninges (pia-arachnoid), and the collection of numbers of small mononuclear cells, probably lymphocytes, in the perivascular lymph-spaces of the leptomeninges. The virus may specifically attack the nerve-cells, but from the evidence now at hand it seems that these cellular aggregates advance along the vessels, like cholera, mechanically obstructing circulation, and that pressure and edema cause the degeneration of the cells. That this aggregation is great enough to accomplish this result is shown by the fact that if a cord is examined from a person dying in the acute stage, it will feel hard to the touch. That the vascular avenue is important is shown by the well-known fact that the cervical and lumbar enlargements are the most constantly affected, and that the anterior horns are more affected than the posterior, the gray matter more than the white, thus exactly following out the order of the most abundant circulation. There are very certainly three factors, viz., cellular exudate, hemorrhage resulting from lesions of the intima which may act mechanically, or by a toxin, or by both influences, and edema. All grades of destruction have been found. If pressure has been slight, or absorption soon takes place, the cells rapidly recover, but if the pressure and anemia have been excessive, the cells go on to necrosis, then polymorphonuclear neurophages in numbers will invade the cell, clear out the necrotic material, and after this connective-tissue overgrowth takes place. In some severe infections hyperemia, perivascular infiltration, hemorrhage, edema, and a diffuse cellular infiltration are found, most pronounced in the anterior horns, but in addition to this, diffused through the white and gray matter of the cord. In these cases, nerve-cells may be entirely absent in the areas affected. The same set of conditions, in a minor degree, is found in the medulla, pons, and the brain. Lesions of the cerebrum or cerebellum large enough to produce motor symptoms are exceedingly rarely found, and while many changes are found in the medulla and pons, their effects do not seem to be constant, as bulbar paralyses may exist without the expected findings at the necroscopy, and vice versa. The effect produced by such vascular changes upon cell-areas is illustrated by the experiments of Rothman. He tied the aorta of dogs below the mesenteric arteries, and found that after 6 hours the cells of the anterior horns began to degenerate, after 12 hours they had become colorless, and had lost their protoplasmic processes. After 4-5 days they had begun to recover, and in 16 days the majority of the cells had recovered their normal appearance. Williams states that degeneration begins in the human subject in 3 days, becomes decided by 10 days, while regeneration is a matter of months.

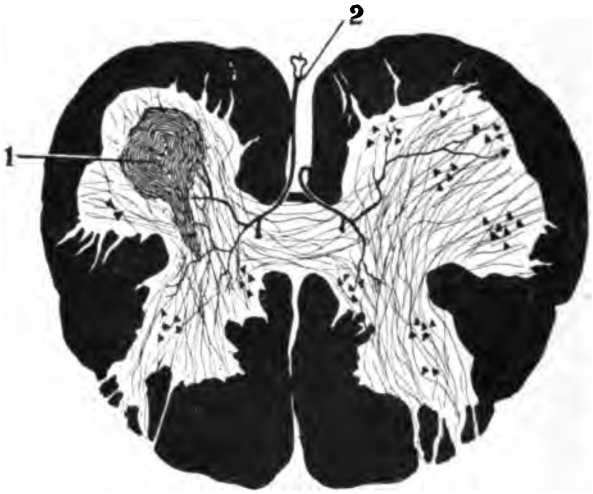


FIGURE 62. ANTERIOR POLIOMYELITIS ACUTA.

1. Indicates the area of softening (observe the blotting-out of motor cell-groups). 2. Indicates an anterior spinal artery (observe that one artery goes to one side and another to the opposite).

Changes in the posterior root-ganglia are practically constant, and may account for the pain which has been experienced, or this may arise from the cellular infiltration which is found along the nerve-roots. Practically as constant as the changes in the nervous system is the reaction of the lymphoid tissues, and the parenchymatous organs, which throughout the body appear to react to the virus. Peyer's patches in the intestine, and the mesenteric lymph-glands show the most marked and acute swelling; the mucosa over Peyer's patches is however unaffected. Generally in parenchymatous organs cloudy swelling is encountered, but in the liver necrotic spots, variable in number, may be found, not always in relation to blood-vessels.

The disappearance of the initial paralysis is probably due to the absorption of the primary edema. Many of the cells at any particular level are slightly affected, if at all. They rapidly regain their functional ability. The extent of the paralysis gives little idea of the extent of the lesion. Sporadic cases rarely have any involvement of the meninges. In every case where the cord is affected there is some degree of inflammation in the bulb, although no symptoms may be present. In cases which died from respiratory failure, an examination of the bulb showed an increasingly intense inflammation extending into the basal ganglia, and yet there was no other symptom than a paralysis of the motor nucleus of the hypoglossal and glossopharyngeal nerves. Muscles deprived of their trophic stimulation lose their function, and lipomatous tissue developes. They may be destroyed, or only weakened. Strauss states that if they are not destroyed they tend to regenerate, this tendency continuing until the 7th to the 9th month, after which no improvement can take place, but a patient of my own showed regeneration after forty-one years of atrophic status. If there is a lack of muscle-tone, the muscle stretches like a rubber band. The muscle may not be strictly paralyzed, and yet be useless from stretching. In such case shortening the tendon will be of service. Relief from apparatus is slower than from surgery. Lipomatous muscles can be restored to function only by grafting them on to healthy muscles by some method of tendon-transplantation. Muscles so to be united should, if possible, be similar in function. The tibialis anticus is peculiarly vulnerable. In all muscles which are apparently completely paralyzed islands of normal tissue may be found, and on the other hand, spots of degeneration may be found in muscles which appear to be perfectly normal, but are innervated from the affected spinal area.

SYMPTOMATOLOGY. Certain facts should be fixed in the mind before we discuss the general symptomatology. In the first place there is probably a pathological difference between the sporadic and the epidemic cases, the lesions in the sporadic forms being limited to special parts of the spinal cord, whereas in the epidemic form the inflammation is liable to be generalized, involving not only the whole spinal cord, but the brain as well, and the pia over both of these structures. Berg suggests that the epidemic form be styled Epidemic Meningomyeloencephalitis. Wickman divides it into eight forms, viz., Spinal Poliomyelitis; cases simulating Landray's Paralysis; Bulbar and Pontile forms; Encephalic form; Ataxic form; Neuritic form; Meningeal form; Abortive form. The cases simulating Landray's Paralysis are in the main instances of the spinal type, in which the process advances usually to an end in death from paralysis of the respiratory organs.

Holt says that this is a poliomyelitis of a severer, and as yet unidentified form. Mueller's grouping is considered more practical, as he attempts to adhere to an anatomical basis. Spinal, Bulbar, Cerebral, and Abortive are his divisions. In considering cases which result in paralysis a pathological-physiological basis is the better one. First: Those cases in which the upper neuron is primarily affected—the Cerebral form of Mueller. Second: A large group where the lower motor neuron is affected—the Spinal and Bulbar forms of Mueller. Some few cases are due to lesions in the cortex, when more of the cortex is involved than is generally the rule; other cases, the majority, seem to depend upon the involvement of the pyramidal tracts, either higher up, or in the cord where that class of cases originate in the Polioencephalitis of Struempell. These cases are uncommon, but such cases do exist as is shown by the occurrence of spastic cases in the same house with the typically flaccid ones. The diffuseness of the pathological process makes any classification open to objection. For instance, a certain number of bulbo-spinal cases may develope an ataxia; this depends upon a lesion in the upper motor neuron, in the brain, or cerebellum, in the pyramidal tracts, or in Clarke's column.

INCUBATION. There is a period of incubation of from 5-7 days, with an extreme limit of from 2-30 days. Following this there are well-marked stages, viz., the prodromal, the paralytic, and the stage of recovery. The prodromal stage may be entirely wanting, as in those well known cases where a robust child goes to bed well, and awakens a paralytic, but such cases are almost absolutely limited to the sporadic type. The average duration of the prodromal stage is from 2-3 days, but exceptionally it may be prolonged to a week, and has been known to last for 3 weeks. The paralytic stage lasts for from 1-3 weeks when it passes off, and the patient is discovered to have a selective paralysis of a limb, or a few muscles of a limb, which rapidly atrophy. The stage of recovery continues during a period up to 7-9 months, after which it is usually considered that further improvement is improbable, but this point will be discussed more definitely in the paragraph on prognosis.

The three important invasion symptoms are a considerable hyperesthesia (over the entire body, or a part of it is an aid to diagnosis), abundant sweats, and a leukopenia, a diminution of the white cells below 7000 (Muller). Most authorities agree that an early and extreme physical prostration, with a weakness of the muscles, or entire limbs, with diminished, or lost reflexes, is the most conspicuous symptom. There is restlessness, peevishness, or apathy with a clear sensorium, except in cerebral cases. In London, in 1910, about 10% showed cerebral symptoms, while only about 4% showed a simultaneous involvement of the brain and the spinal cord. After this restless period there is a fever of 103° or higher, which may last a fortnight, and by its persistence might suggest typhoid, especially when we remember that it is combined with great physical prostration. This fever at times shows a recrudescence after being absent for a week or two, generally with the advent of bulbar symptoms. A chill is rare, but sweating is very common, and also the hyperesthesia mentioned above, so that the child cries if touched. Diarrhea and gastrointestinal troubles are possible, but were more common in the European epidemics than in the American; in Krause's series in Westphalia it was 70%, while in New York it was only 25%. There is a loss of appetite and nausea, but persistent vomiting is rare. One of my cases gave a history of vomiting for three days. The tonsils are affected at times, but they are rarely reddened, there is never an exudate, and on the whole, symptoms in the respiratory tract have been conspicuous by their absence. When convulsions are persistent it indicates an involvement of the cerebral cortex, and is a rare condition. Muscular twitchings, and tremors show that there is an irritation of the cord, and they are accompanied by a weakness of separate muscles, or of the limbs in general, without change in the deep reflexes. Spontaneous pain is rather transient, and located in the back of the head, in the back, the neck, quite often in the legs, and in the muscles to be paralyzed. It is probably due to the inflammatory reaction in the posterior root-ganglia. No percentage would be reliable, on account of the extreme youth of the patients. Pain, both spontaneous, and that produced by motion, tends to increase as the acute stage approaches. There are two types of developement of the disease in this period. Generally there is a gradual increase in the severity of the symptoms; on the other hand, we may find in two or three days a total cessation of all symptoms, an apparent recovery, and then a relapse into paralysis. The severity of this initial stage is exceedingly variable, and bears no relation to the severity or extent of the subsequent paralysis or course of the disease.

Paralytic stage. It is a peculiar fact that the advent of paralysis is more retarded in adults than in children, among whom it comes on in 2-3 days, and also in its extension from one group of muscles to another. After the subsidence of the fever no extension of the paralysis is to be looked for except in very rare cases, in which there is a recrudescence of the fever after 2-3 weeks, and in such cases it usually signalizes an involvement of the bulbar nuclei. There are cases, however, where there is such an oscillation of the temperature that after 2-4 days of apparent fall, the rise is very distinct, and is followed by an increased area of paralysis. This sudden fever then, in recapitulation, is followed by a general, or localized paralysis, almost always of the flaccid type, with the reaction of degeneration, and an early atrophy in the muscles permanently paralyzed. The paralysis is most commonly of one or both legs, but may occur in any part of the body. While there is often a rigidity of the muscles of the neck and the spine, it soon gives place to a flaccidity of the muscles, and the patellar reflex is lost in most cases. This rigidity of the neck and spine may manifest itself from the 1st to the 3rd day of the prodromal stage. The gravest possible sign is the disappearance of both the galvanic and faradic response, since it shows that there has been a very definite degeneration of the peripheral nerves, from their nuclei in the cord to their terminations in the muscle-fibres. Under such conditions the muscles undergo a lipomatous change, and in many cases cannot regenerate. The tibialis anticus is the most vulnerable of all the muscles.

Lovett and Lucas found that the paralysis of one leg is nearly four times as common as the paralysis of both legs, while the paralysis of an arm and leg of one side is more common than a crossed paralysis, i. e., an arm of the one side, and the leg of the other. The internal muscles of an extremity are more frequently paralyzed than the external, and the anterior more often than the posterior. The most common muscle to be affected in the leg is the quadriceps; the next in frequency are the tibialis anticus, and the anterior muscles of the lower leg. If the hamstring muscles are affected, it is more often the internal than the external, and the sartorius is frequently unaffected when the quadriceps is paralyzed. The internal rotators of the thigh are more frequently affected than the external, and the adductors more often than the abductors. The short toe-flexors they found were least liable to be affected. In the upper extremity the arm is more frequently paralyzed than the forearm, and the deltoid was the muscle most often attacked. The biceps, triceps and scapular muscles may also be affected.

Paralysis of the muscles of the neck and back were generally associated with some other lesion. Diaphragmatic paralysis is usually unilateral, and paralysis of the sphincters is rare, but in the New York epidemic of 1907 was seen among children of from 3-8 years of age. This incontinence has never been reported to have occurred in the sporadic type of case. When the cerebellum is attacked there is an acute onset, occipital headache, ataxia and nystagmus, and yet in many cases there has been a complete recovery. Paralysis of one or two ocular muscles, and a slight degree of facial paralysis has been the only residuum of some attacks. At other times a child who had gone to bed well may wake up with a squint, in three-quarters of the cases the paralysis being of the external rectus, and seems to depend upon a slight focal encephalitis. Meningitis of some grade is always a factor, and may be severe in the early stages, and cloud the diagnosis, since it may cover the real character of the changes; in fulminant cases it is common; it may be prolonged or transient. Bulbar palsies are actually rare, an involvement of the 7th nucleus being the commonest form of what is really a polioencephalitis inferior, and this is an important link in establishing the common etiology of poliomyelitis and polioencephalitis superior. There are two varieties of pain: the first is meningeal, for which there is no remedy; the second is nutritional, and is from the mechanical effect of the anemia of the neurons which innervate the muscles. It is analogous to the pain and tire which normally follow cramped positions, and prolonged exercise of muscles. That this is the real nature of the prolonged pain is proved by the nature of the methods which will relieve them, viz., galvanic electricity, suspension, and support. The atony of the muscles is still further favored by the passive stretching, which also increases the pain. Foerster says that cases may show spontaneous pain in the limbs, and tenderness on pressure, not at first, but in the paralytic stage, and it then may be persistent in otherwise atypical cases. These pains he says are not to be looked upon as due to any especial disease of the peripheral nerves. Pain and tenderness confined to the joints and muscles were common in the 1907 epidemic. In one epidemic sharp pains in the breast were reported, and in other cases in one limb, which was to be the site of the initial paralysis. There may be a skin eruption, both papular and vesicular, all over the body, especially on the lower extremities, both on the anterior and posterior surfaces, and on the dorsum of the feet. It is superficial, lying between the Malpighian and corneal layers, amounting to only a slight perivascular infiltration, and nothing else.

The skeleton may atrophy, due partly to a cessation of trophic impulses, and partly to the absence of a suitable blood-supply on account of a lack of muscular activity. Falling of the hair, chilblains, grooved nails, and hypersecretion of sweat are other occasional trophic results.

The state of general paralysis lasts from 1-3 weeks, and then passes away, leaving the child paralyzed in a few muscles of a limb, as a general rule, although some cases result in the paralysis of a whole limb, or an entire side. These paralyzed muscles will atrophy, the limb as a whole will be blue and cold, and its future growth is stopped or greatly retarded. At this stage there is ordinarily no pain, and if present, it is due to a secondary neuritis, or to an inflammation in the muscle from overstretching, or rapidity of the atrophic changes. Sensibility is well preserved. Paralytic contractures are rather common, due to the action of unopposed antagonists, and we also see scoliosis, lordosis, and loose joints.

Abortive Cases. There are two classes of cases occurring during an epidemic which are liable to lead to confusion. The first are those who, after a typical onset, develope a cerebral instead of a spinal form of paralysis. These are instances of Struempell's paralysis, or polioencephalitis superior. The second class is the abortive type of poliomyelitis. The symptoms of this type are fairly uniform, but yet not sufficiently characteristic to enable one to make the diagnosis when seen by themselves. The onset is usually abrupt, often with vomiting, constipation, great prostration, and a rise of temperature to 101-103 degrees. Accompanying these general symptoms, there is very often hyperesthesia, pains in the neck and back, and in many cases some stiffness of the neck. There is usually enough general prostration to keep the patient in bed. The duration of the attack is generally 2-3 days, after which it rapidly subsides, and the case ends in a perfect recovery. As a proof that these are cases of poliomyelitis, Flexner and Clark have shown that the blood-serum of these cases has the same power of neutralizing the poliomyelitis virus that is displayed by typical cases, both sera having this power from the developement of antibodies. The diagnosis of these cases is of the highest importance, since any hope of the control of the disease rests upon the identification of the abortive cases, and of the carriers, whether recovered cases, or persons who have been in contact with the infection. In the first place, isolation and quarantine are then most efficient, and in the second place, if we can recognize the prodromal stage, prevention of the paralysis is possible then, if ever.

Diagnosis. In the absence of an epidemic, a diagnosis in the initial stage is very doubtful, and is hardly possible except by lumbar puncture. If the case is one of poliomyelitis, the fluid will be found to be under increased pressure it will be clear, or slightly flocculent, and in the prodromal stage it will probably be opalescent, show an increase in protein, and an excess of white cells, principally lymphocytes. Draper and Peabody assert that there is a constant and excessive leucocytosis up to 30,000. After the paralysis has appeared, the fluid, although still increased in amount, will be found to be clear, with a diminution of both protein and lymphocytes from the findings in the initial stage, but still above the normal. In the absence of an epidemic, this will give the only basis for the diagnosis of bulbar, meningeal, cerebral, and abortive forms. There seems to be a preponderance of opinion in favor of a distinction in symptomatology and prognosis between the sporadic and the epidemic forms. The sporadic form is almost never fatal, and almost never marked by complete recovery from the paralysis, while the epidemic form shows a considerable mortality, but a surprising number of approximately perfect recoveries. The lesions of the epidemic form are also much wider in distribution.

The diagnosis rests upon irritability, incoördination, prostration, retraction of the head, spastic condition of the spine, occipital headache, backache, rising temperature, sweat, accelerated pulse-rate, the unaccountable amount of prostration being the striking and determining symptom. This diagnosis must of course be tentative, but it is at this time, if ever, that treatment can be actually curative, if successful. The appearance in 12-36 hours of a general flaccid paralysis makes the diagnosis certain, but too late to do more than attempt to remedy damage already done to the nervous tissues.

Differential Diagnosis. It is a most satisfactory plan in differentiating this form of paralysis from those possessing some degree of similarity to it, to divide them into those which are respectively cerebral, spinal, and peripheral in origin.

Cerebral palsies are strictly hemiplegic in distribution, tongue, face, and speech often suffer; the advent is abrupt, and there is rigidity of the limbs, the tendon reactions are increased, the electrical reactions are preserved, or only diminished in quantity, and there is no loss of faradic irritability. The convulsions, if present, are focal or affecting only one side of the body, in any event commencing locally, even if they afterwards become general. There is no extreme wasting of the muscles. In older children there may be aphasia. There is mental depression of a profound character, and there may be final deterioration. As a late result there is apt to be athetosis or hemiplegic contracture. In anterior poliomyelitis, on the contrary, the distribution of the paralysis is not generally hemiplegic, the tongue, face, and speech are unaffected, the tendon reactions disappear so far only as they are in the area of the muscles involved. There is the electrical reaction of degeneration, and faradic irritability soon disappears. There is rapid wasting of the muscles. A late result is deformity resulting from partial paralyses of the limbs. Mentality is unaffected. If convulsions occur they are general in character.

Atrophic Forms of Paralysis from other causes. If the child has a transverse myelitis there will be a paralysis of sensation, as well as of motion; of the sphincters also; bedsores will occur; there will be no rapid diminution of the area of the paralysis, and there will be a long-continued fever. The electrical reactions will not be so dissimilar as to have any diagnostic value.

Pseudo-hypertrophic Paralysis. Occasionally will be confused on account of the fact that there may be a deposit of fat in atrophied muscles in late stages of anterior poliomyelitis. This increase in muscular size is, however, a late symptom in anterior poliomyelitis, and an early symptom in pseudo-hypertrophic paralysis.

Progressive Muscular Atrophy. Rare in children. The slowness of the disease ought to differentiate it. If this question arises years after the attack, it may be decided on these grounds: if the attack occurred in infancy, or early adolescence, a Progressive Atrophy would first be displayed in the face, which is almost never the site of the atrophy of Poliomyelitis. If the paralysis and atrophy occurred in the limbs, its rate of progress would be diagnostic, since Poliomyelitis produces its effects in days, or at least weeks, while Progressive Muscular Atrophy demands months and years of time. Objectively, the carved-out appearance of the atrophied muscles, bounded by well-developed ones, differs from the more general paralyzed condition of Anterior Poliomyelitis.

Hemorrhage into the Anterior Horn. This is a rare accident, but the results are identical, and it cannot be differentiated, except possibly by the history, but that is conclusive, since there is a sensation of a blow upon the spine, and paralysis is instantaneous, instead of a developement of hours, or generally of days.

Cerebro-spinal Meningitis. The history of the attack is dissimilar. If there are convulsions they may be general, but the paralysis may be of the cerebral type, that is spastic, instead of flaccid; the spinal and nuchal rigidity is peculiar to it, and an extensive degree of paralysis will not occur without a meningitis, which will give rise to far more pain than is found in anterior poliomyelitis.

In general it may be said that all diseases of the spinal cord producing paralysis, but not affecting the anterior horns of gray matter, are to be distinguished by the absence of reaction of degeneration, and of atrophy and fibrillation; the paralysis is spastic as are the contractures; there is an increase in the reflexes, and mentality is liable to be decreased.

Multiple Neuritis. This is peculiarly peripheral. It is symmetrical in affecting both sides of the body; it has marked sensory disturbances, in that, aside from spontaneous pain, and changes in sensation in the extremities, the nerve-trunks are swollen and tender, and there is a history of intoxication or infection, and wrist drop and foot drop are diagnostic, if present. There are, however, conditions favorable to error, since the patient may be too young to give us any criterion of his sensations. It is also true that polyneuritis may complicate anterior poliomyelitis, but it may be said that toxic neuritis is rare in children, acute anterior poliomyelitis extremely rare in adults.

Rheumatism. Since both diseases commence with a moderate degree of fever, and often result in a speedy immobility of the limbs, and patients may be so young as to render doubtful the cause of this immobility, errors are sometimes made. Careful examination will disclose that the child *can* move the limb, but is *unwilling* to do so, if the cause be rheumatism. It will also be found that the limb is hot, in place of the coldness of the spinal paralysis; that the joint is tender to pressure; that instead of a blue coldness of the skin there is sweating, but no atrophy is present, which is to be expected in an early stage of anterior poliomyelitis. After some inflammatory conditions of joints secondary atrophy does take place, but the patients are generally adults, and the symptoms localizing the trouble in the joints, and not in the spinal cord, are usually convincing.

Congenital Dislocation of the Hip. May be taken for a late case of anterior poliomyelitis, but careful examination of the hip joints will show the anatomical faults, the gait is peculiarly diagnostic, and there are none of the positive symptoms of anterior poliomyelitis.

Coxalgia (Hip Disease). In the first two days of anterior poliomyelitis we may have pain in the back, the leg may be flexed and rotated outwardly, and it may be immobile. This would remind one of hip disease, but an examination will show that the articulation is perfect, and that the leg is capable of passive, but not of voluntary motion.

Erb's Palsy. This may appear like a case of anterior poliomyelitis affecting the cervical enlargement, but the patient will be a new-born infant, and there will be a history of traumatism during labor. Examination will show that it is a localized neuritis of the brachial plexus. There will be a localized anesthesia in the distribution of the circumflex nerve, that is, over the lower deltoid region, and the deltoid, biceps, coraco-brachialis, and supinator longus are paralyzed.

Rachitis and Marasmus. The two conditions are similar in an unwillingness to walk, sudden onset of fever, and pain and tenderness in the limbs in children. There is no history of a preceding illness. They may complain of a certain degree of pain, or they may lack those symptoms entirely. If the cases are carefully examined, it will be found that cases of anterior poliomyelitis have no tenderness of joints, though perhaps some from muscular spasm, and that the legs are paralyzed. In the case of rachitis we shall find that there is no paralysis, but that the child is unable to walk on account of the softness and the tenderness of the bones, especially about the articulations. The pain and tenderness in rachitis is not limited to a few muscles, but is in the bones, as has been said, and generally through the muscles. The gums are tender and swollen, and there is sweating of the body. Such a child may have been brought up in the best of surroundings, but investigation will reveal a defect in the nutrition. There is another group which is marasmatic, and the difficulty in locomotion is, in this case, due to a weakness of the muscles.

PROGNOSIS. Danger to life from this disease was formerly considered to be slight and it is fairly true of the sporadic cases seen formerly, though the history of epidemics shows that sporadic cases are rarely fatal, but generally leave a large degree of permanent paralysis, while the epidemic form carries a considerable fatality, yet a larger percentage of the paralysis is curable. Flexner stated that in epidemics there was a death-rate of from 5-10%, and that 75% were paralyzed to some extent. In the epidemic in Nebraska, only 25% suffered paralysis. Holt says that taken altogether the mortality is 12% on the average, and much lower in sporadic cases. The prognosis as to life is better in children than in later years. The mortality was 12.2% in cases under the age of 12, while it was 27.9% in the years from 12-30. More children die on the 4th day than on any succeeding one, the death-rate rapidly declining on the succeeding days, and the prognosis for life being good after the first week. The probable extent of the paralysis is estimated thus: a halt of 24-30 hours indicates almost certainly that its full extent has been reached. Rarely, after a week or two there has been a recurrence of the fever, and an extension of the paralysis. This has been ascribed to an invasion of the bulb. The estimation of the permanency of the paralysis is not now considered to be possible to the extent that obtained before methods of investigation were as accurate as at present. Many more cases recover than was thought possible. There is no disease in which faradic irritability is lost so soon, and the galvanic reaction is so difficult of demonstration, and is so changed in quality. It is now acknowledged that electricity has been overrated as a criterion of the permanence of the paralysis. A return of the faradic response indicates a return of voluntary motion, and by as much as this is early, by so much greater will be the completeness of the recovery. This is the only positive electrical criterion to which all subscribe, and the converse does not prove to be equally true. It is generally agreed that an immediate loss of the faradic response indicates an incurable paralysis, but if this loss is delayed until a week after the advent of the paralysis, the muscle will probably regain its power, and this is almost certainly true if the faradic response is not lost for two or three weeks.

How long can faradic response be absent and yet recovery finally take place? Here is a wide diversity of opinion. Gowers said that an absence of 10 days indicated a permanent partial paralysis, and after an absence of this response for 3 months very little improvement could be hoped for. On the other hand Sinkler quotes a case where it was absent for 15 months, and after 60 days of active treatment it returned with voluntary motion. Stern reports a case of a child of 23 months of age who was paralyzed at 4 months, and was markedly rachitic. The deltoid and biceps were weakened, and the extensors of the back and legs. Voluntary adduction, flexion, and inward rotation of the thigh, and the power to extend the legs was lost, but the other motions were only slightly weakened. The legs and feet were cyanosed and cold. The paralyzed muscles did not respond to the faradic current, and the galvanic current produced only slight and sluggish contractions. After 9 months treatment, all the muscles responded to strong faradic currents, in 12 months the patient could sit erect, and walk in braces, and in 13 months the patient could walk erect, and innervate all his muscles. I have seen a case with Dr. John Hutchinson, of this city, which after 41 years as a cripple, and loaded with braces, has recovered good power of locomotion, and is showing restoration of function in most of the paralyzed muscles by the use of corrective shoes for the talipes varus resulting from contractures, and well-selected homœopathic remedies for the weakened spinal cells.

Muscles will respond to galvanic currents before they are affected by faradic, and the slow wave of the sinusoidal at an earlier period than to interrupted galvanism. The advent of the reaction of degeneration, i. e., anodal closure contracture of a galvanic current being equal to or greater than cathodal closure contracture, has been considered to be the death-blow to any hopes of recovery. Lezinsky says that it is not an infallible guide, since all that it actually shows is that there is an interference with the nutrition of the motor tracts. The conservative opinion is that if it is developed later, there is a probability of some repair in the muscle, but if it is developed within ten days, entire regeneration of function is improbable, and permanent paralysis of the muscle is almost certain. Hoppe reports a case where it was present at the end of two years, and nevertheless the patient recovered almost complete control of the affected muscles. In electrically testing muscles, the indifferent pole should be 4x6 inches in area, and well moistened, while the pole applied to the muscle should be a point 1 centimetre in size, and the current necessarily should be strong enough to cause some pain.

Paralysis of the muscles of the neck is rare, but is usually recoverable, and paralysis of the diaphragm is still rarer. In the process of recovery it has been established that a muscle will respond to a volitional impulse before it will react to faradism. As a prognostic test, Regnier advances a method which he terms negative contracture or resistance test. .He calls the effort to raise an arm a *positive* one, to hold it in that position a *negative* one. Another form of test would be the contraction of one group of muscles, while trying voluntarily to contract another group. The test is simple. A limb is raised to a determined height, and held there, and at the same time firm pressure is made upon the segment of the limb to be tested, and as near the periphery as possible, the pressure being exerted as nearly as can be upon the tendon. The muscles of the extremity are now made to contract, which brings out the reflex negative contractability of the injured member, the intensity of the contraction being in proportion to the excitability of the motor cells. It was found to be present in some measure in more than 50% of all cases, even when reaction of degeneration was present. A method of ascertaining the muscles actually deficient, and their degree is to be found in an article by Lovett and Martin, in the Jour. A. M. A., Mar. 4th, 1916. Hopkins believes that the following test may be relied upon. If on voluntarily opening the fingers, spasmodic movements occur, mechanical methods will be only partially successful. If when the patient is asked to extend the fingers, he can do so only by simultaneously moving the fingers of the other hand, the case is hopeless; if, however, on being asked to move his fingers, they respond by the slightest quiver, and the surgeon then further contracting his hand shall ask him to repeat the act, and the patient succeeds in moving them back to the point whence the surgeon started them, such a case, should it have lasted one year or thirty, will end in complete recovery.

There is some tendency in later life to develope some form of muscular atrophy, or dystrophy, from the damage to the cells of the anterior horn. Multiple sclerosis has also had such an origin.

TREATMENT. The treatment of this disease must be divided into three divisions, viz., treatment of the invasion, of the acute stage, and the stage of paralysis. The actual damage to nervous tissue occurs in the stage of invasion, in the first hours; hope of cure therefore rests upon the recognition of the disease in the stage of invasion. Sporadic cases must, in the vast majority of instances, elude the diagnostic powers of the physician, but if a child with catarrhal symptoms displays a muscular prostration out of all proportion to the illness complained of, it should put the physician upon his guard, and a lumbar puncture should be done, when the discovery of an opalescence of the cerebro-spinal fluid will make the diagnosis sure. The patient and attendants should be isolated during the acute stage, i. e., for three weeks after the appearance of the symptoms. The secretions of the patient should be sterilized at once by boiling or disinfectants. The nose and throat, not only of the patient, but of the attendants also, should be sterilized by sprays and gargles, hydrogen peroxide 1-3% being an efficient one among others. Other sprays recommended are colloidal silver, argyrol 25%, protargol 30%, chinosal 1-2000, applied postnasally. The room should have a terminal disinfection by scrubbing, fresh air and sunlight. In an epidemic there is less excuse for error, and under such conditions every catarrhal case showing notable loss of muscular power should be at once put into bed, upon his side or an inclined plane, never upon the back if the bed is level. Lumbar puncture should be done at once, and if the fluid is opalescent quarantine should at once be established, and the sterilization of the nose and throat inaugurated. Free movements of the bowels assist the drainage of the spinal canal, and therefore, whatever be the therapy, it is best to give a cathartic by the mouth, castor oil is the best, and olive oil by the rectum. At this stage Belladonna and Gelsemium are most often indicated, Aconite less often, and Cicuta as well as Belladonna when severe pain, opisthotonus or convulsions show an early involvement of the meninges. Can the microbic activity be checked at its source? Cushing and Crowe showed that if urotropin was taken by the mouth, it was in part excreted into the subdural space, and Flexner demonstrated that if a large dose was given by the mouth, that it could be discovered in the spinal fluid. It was also shown that if urotropin was administered to a monkey, and it could be demonstrated in his spinal fluid, and the monkey was then innoculated intra-cerebrally, and the drug afterward administered by the mouth, that a proportion of monkeys so treated would show an incubation period of 24, instead of the customary 6-8 days, and further it was found that the onset of the expected paralysis was entirely prevented. Preble reports two human cases, seen soon after the advent of the paralysis, who received large doses of urotropin with the result that the paralytic stage was cut short, and the cases made good recoveries. In view of these reports it is advised that urotropin be administered at once in doses of 2 grains every 6 hours to a child of 2-3 years, 3 grains at a dose to children of from 6-10, and 5 grains to an adult. Personally I have not seen any beneficial effects from the administration of urotropin when administered after the invasion period, and have not had the opportunity of observing its effects when administered in the stage of invasion, but if a case should be discovered thus early, I should have high hopes of its value, since there is a demonstrated scientific basis for its use, and apparently it only awaits a developement of technique to make it an efficient treatment at the only period in the disease when cure can be hoped for. Few cases, however, are seen at this stage, since the patient when first seen is usually a febrile paralytic. It has been claimed that hot baths, and hot enemas of magnesia sulph. would abort an attack.

Acute Stage. The treatment of this stage has been very diverse. One plan has been most energetic, while the simple conservation of the comfort of the patient has had equally authoritative endorsement. The active treatment consists in a very vigorous counter-irritation to the spine, perhaps by the Pacquelin cautery, or ice-bags may be applied the whole length of the spine. The child should be immobilized by a cast extending from the toes to the free ribs. Fever and pain are to be antagonized by a free use of antipyretics, and analgesics, or narcotics. As soon as possible the muscles are to be stimulated by electricity and massage. Luisada claims that if electrical treatment is begun as soon as the fever subsides, recovery will be quicker, and more complete than if its use is delayed. He advises that the indifferent electrode should be placed above, or below, the affected centers in the spine, in order that the current shall not pass the inflamed portion of the cord. Williams says that galvanism should be used in the first week, and that those who object to its use do not understand its effects. Gibney says that so soon as the diagnosis is established the limb, or limbs, should be placed upon a wire, iron, or trough-splint, well-padded, with the feet at right angles with the legs, knees in slight flexion, and the thighs in line with the long axis of the body. This is necessary to prevent the fatigue, or strain of muscles by allowing the child to swing a dangling limb, or bear weight upon a weakened joint. Limbs are also liable to become deformed, not only from contractions, and later by contractures, but also by falling into false positions while the muscles and joints are too weak to bear their weight. The more conservative plan, and the one which I have been accustomed to employ, seeks to reduce interference with the comfort of the patient to a minimum, but since prevention is far more certain than cure, I believe that one should always be on the watch for the first signs of deformity, and should combat them by effectual means, no matter how cumbersome. The remedies to be employed are stated at the end of the article. The patient should be guarded from noise or interference, although many may be amused by toys and reading without injury. The attendants should be few, the diet milk and cereal gruels and the bowels should move freely. We should never forget that the circulation in the affected limbs is deficient, and wrapping them in a light woolen shawl is often the best treatment. In a severer paralysis they should be wrapped in cotton, and supported by a flannel roller, and the weight of the bed clothes should be carried on a cradle to avoid the production of drop-foot. If there is a positive tendency to this deformity, or to eversion of the feet and legs, it will generally be effectively prevented by the use of sand bags, but it may require some form of cradle-splint or plaster-cast. In all cases the support should be removable, so that the limbs may be gently massaged from the earliest period. This is the only period, generally speaking, when pain is a troublesome symptom. Hot cloths to the limbs, rubbing them with the warm, well-oiled hand, ice to the head, or if these measures are not effective, a bath at from 95°-105° for fifteen minutes, will often be more efficient than drugs. Stroking application of a mild galvanic electrode, or with warmed flannel also has its value. In giving a bath, three conditions should be fulfilled, viz., the depth of the water should not be so great as to impede respiration; the temperature of the water should be constantly maintained at the initial temperature; the exposed parts of the body should be kept warm. Mitchell states that the removal of an ounce and a half of spinal fluid by lumbar puncture has relieved intense pain and dyspnea. Before dismissing the subject of pain, it must be pointed out that it is from two very different causes. Pain in the muscles and articulations may be relieved by the measures just detailed, but the other variety, lancinating, and accompanied by muscular spasm is from irritation of the nerve-roots, or involvement of the meninges. This pain can be relieved only by lumbar puncture, or by narcotics, or analgesics. Deodorized tincture of opium has been advised as the best drug, given in doses of a drop an hour even to a young child, a less dose to the very young, while a child of ten years will tolerate a larger one.

When the fever has departed, and the muscles have lost their tenderness, or become tolerant of handling, the problem has resolved itself into the treatment of a case of paralysis with more or less deformity. If the site of the lesion has been spinal, the paralysis will be flaccid, and lower neuron in type, while it will be spastic to the extent that the upper neuron and the brain have been implicated; there is often some mixture of the two types. In the treatment of this stage there is not a unanimity of opinion, but the general opinion is that massage, electricity, braces, nerve and muscle-transference are necessary, to a variable degree, yet in a large proportion of cases simple measures, patiently employed, will produce good results.

Massage is commended by all; by some said to confer all the benefits hoped for from the use of electricity, and when combined with resistive movements and educational gymnastics will conserve the muscles, and stimulate their growth without frightening the patient. It will also, in most cases, obviate the necessity for the use of braces, or the employment of operative procedures. The massage should be light, as vigorous massage overstrains weak muscles. The massage should be given on a firm table, all clothing having been removed, and the mind of the patient should be concentrated on the work. When the nervous mechanism has been but slightly injured, recovery is prompt, but in some cases there is Apraxia, i. e., the nervous mechanism is present, the power of muscular contraction is present, but the coördination of the two seems to be lost. If in such cases the patient, while undergoing passive treatment, tries to make the movement himself, he will develope the association-paths anew. Even before resistive movements are well-tolerated, the muscles should be aided to regain their function by placing the child in a bath-tub moderately filled with water at 100° F., made saline with sea-salt to increase the specific gravity, and at the same time to stimulate the skin. With the limbs supported by the water, and the child tempted to movements of them by the presence of floating toys, it will be found immediate improvement will be noticed, since muscular movements can be aroused earlier by volition than by electricity, or any other external stimulant. Where financial conditions have made massage unattainable, I have directed some person in the family to follow the bath with a daily rubbing of the muscles with the oiled hand for about fifteen minutes. The muscles should be rubbed in an upward direction, following this by an alternating compression and relaxation of the muscle-belly. A muscle-beater, made from a rubber ball and a handle improvised from wire, is also a good substitute for massage. Muscle-training should not be started until all acute symptoms have subsided, and the muscles have lost their irritability, probably in a month or six weeks after the attack. Resistive movements should be for from twenty minutes to a half-hour every day, or other day. Muscle-training is best accomplished when it takes the form of play for young children and competition for the older ones. Resist the temptation to get the patient into braces or to walk too early; heavy braces are apt to mean the end of developement. It is best for a child to creep first, kneel next, and then to try to walk.

Electricity has been a storm-center as many say that its employment is time wasted, while others as strongly insist upon its superlative efficiency. If improvement of nutrition in muscular tissue were the only question at issue, the argument that massage perfectly replaces it might be considered valid, but it seems fairly certain that its effects are deeper, that it stimulates the whole neuron, and therefore must increase the nutrition of the cells in the sphere of its application. Static electricity will evoke the first response, but it is generally unavailable, while the sinusoidal current is next in potency, and may be available. The utmost care should be exercised that the contractility of the muscle shall not be exhausted, and therefore four or five contractions are all that should be demanded of each muscle until muscle-contraction has become a routine matter. Usually galvanism will be the practicable method, and the indifferent electrode should be large, and kept moist, so that proper contraction may be produced without injuring the sensitive skin. As soon as the muscles will respond, the faradic current may be used. In using electricity it should be impressed upon the patient that an aggravation may result, and after a remission improvement will begin on the resumption of the treatment. It is a good plan to follow the treatment with a hot bath, and massage enough to fill a thirty minute period with the whole treatment. Potpeschnig begins with faradism and massage, two or three weeks after the acute process has begun to decline, and as the deltoid is the muscle most generally affected, to prevent its overstretching, he has the patient sleep with the arm over the head at night.

The use of orthopedic apparatus is advised by some as one of the very earliest curative measures, and postponed by others until deformity shows signs of decided increase. The point upon which the whole question ought to hinge is this: are the weakened muscles being overstretched by their antagonists, or by the weight of the limb? Mild cases are better without them since a brace is a splint, and tends to weaken a muscle by disuse ot it, and also adds weight to be moved. Therefore, so long as a muscle tends to improve, even slightly, and an improper position tends to improve, the limb is better without the brace. If increase of deformity becomes evident, an orthopedic shoe, a rubber muscle, as described below, or at the most a brace up the leg to the knee may remedy the condition. In paralysis of the leg, the brace should never be extended above the waist, unless it is absolutely demonstrated that it is necessary. If contractures have taken place, they must be corrected, and all other deformities, before the patient can stand on his feet and walk. Tendon grafting, anastomosis, or transfer are often beneficial and often present the best method of assisting the patient, but nerve-anastomosis has not as yet justified its promise. Schwab and Allison isolate the groups of muscles which produce the contracture, or deformity. This offending group is then cut off from the central nervous system by isolating the nerve innervating these muscles, and injecting it with alcohol. Afterwards the antagonists are developed by massage and educational exercises. Anchylosing operations (arthrodesis), especially about the ankle, are sometimes demanded when joints are so relaxed (flail-joint) as to make the limb useless for support or progression. Under such conditions they may be said to cure the patient, so far as ordinary activities are concerned. This operation should not be done under the age of nine years, as previous to that the bones are too soft to produce a firm and rigid union.

Rubber muscles often serve a good purpose. Sometimes all that is needed is a light shoe, to the toe of which is attached a common rubber band. This in turn is fastened to a cord fastened about the leg at the knee. A more elaborate method is that of Meisenbach. He uses strips of rubber dam, of a strength proportionate to the work. It is cut into strips from 2-4 centimetres wide, and from 12-24 in length. At the end of the rubber strips pieces of zinc oxide plaster are attached, which grip the ends of the rubber to the extent of about 4 centimetres, and extend 10-12 centimetres beyond. It should grasp the rubber on all sides, and be compressed in a vise, or pressed with a mallet, so that it shall not break away. It may need slight heating. It is most applicable to the ankle, knee, wrist and shoulder. One end of the artificial muscle is applied over the joint, corresponding approximately to the origin of the muscle, and the other end to its insertion. For instance, in the case of the peronei, one end is applied to the outer border of the foot, passing over the cuboid, and the other end is applied to the outer side of the leg, midway between the ankle and the knee. If the tibialis posticus is involved, its origin and insertion are guides to its application. In every instance it is applied as taut as the treatment warrants, but if the limb is held in a proper position, little tension is needed to retain the limb. When applied to the wrist, or other places where more than one muscle is involved, the plaster may be cut in a Y, or other form as needed. This treatment supplements the lost power until the muscles have recovered, the force stimulates the normal better than any other method, the contraction of opposing muscles is prevented, the rubber over the affected muscle causes a local hyperemia, manifested by the perspiration under the rubber, it does not immobilize the point, nor interfere with passive motion, massage nor electrical contraction, its action is continuous, and does not interfere with bathing, or the clothing.

THERAPEUTICS. In the first stage, if the patient has fever, especially if there is tingling of the extremities, Aconite; but discontinue it with the first appearance of paralysis. If the fever is very high, and sudden in onset, with delirium, and continues after the advent of paralysis, Belladonna should be considered. If it succeeds an infection, and the patient has feeble pulse, low fever, or a reduced temperature, and a tendency to ptosis, Gelsemium. Clinically, Gelsemium has the greatest amount of testimony to its efficiency in the initial and intermediate stages. If in a case succeeded by some other infection, we have symptoms of great restlessness, and with it muscular soreness, we should think of Rhus tox., or Arsenicum, and sometimes of Lachesis. With a hard bounding pulse, or on the contrary a feeble but rapid one, with much cerebral excitement, Veratrum vir. If spinal symptoms are persistent, and muscular jerkings are a feature of the case, Cuprum. In the intermediate stage Phosphorus may be of value, on account of its appropriateness in any atrophic condition. Cobb, of Chicago, advises Phosphoric acid in the intermediate, and often in the late stages, as a remedy of great promise. Mercurius may be indicated, since its action is on interstitial tissue, and poliomyelitis is a disease where there has been an interstitial reaction after a vascular occlusion. Secale has been advised, and in this case has been found to be of greatest value when used in tangible doses, viz., ten drops every four hours for a child of two years and below, and an increase of two drops to the dose for every additional year of age. In late cases Baryta carb., and Causticum, and Graphites have been advised, the latter on account of its action on scar tissue. It is worthy of attention that Felix mas has produced symptoms very strongly resembling acute poliomyelitis (Magnus Levy, Berl. klin. Woch., Mar. 25th, 1911).

LANDRAY'S PARALYSIS.

Definition. It is an acute parenchymatous degeneration of the peripheral motor neurons, of toxic or infectious origin, which process gives rise to an acute ascending motor paralysis. It may be a form of poliomyelitis.

Symptoms of a Typical Case. There is a rapidly developing flaccid paralysis, involving the whole body, and requiring from a few hours to a few days for its full evolution. It begins in the lower extremities, and rapidly extends upward, involving successively the feet, legs, trunk, arms, face, and the muscles innervated from the bulb, particularly those of the diaphragm, heart, and pharynx. The sphincters are not involved, the deep and superficial reflexes are both abolished, there are no sensory disturbances, and the mind remains clear, the case generally ending in death within a week.

Differential Diagnosis. This is from Meningeal Hemorrhage, Peripheral Neuritis, Anterior Poliomyelitis, Myelitis, and Periodic Family Paralysis.

Age. It is a disease almost exclusively of persons between the ages of twenty and forty. It has been noted in children.

Sex. With few exceptions it is confined to the male sex.

Etiology. It commonly occurs in the colder months and refrigeration seems competent to be the sole cause in some cases. The author observed a case which, from his intimate knowledge of the patient, and his conditions, could be attributed solely to over-exposure, combined with over-exertion, during the blizzard of 1888. Another case occurred when, after a debauch, the "Keeley Cure" was invoked for the third time. It has seemed to result directly from alcoholism, and much more often from injuries received while under the influence of alcohol. It has followed the infections during convalescence. It has succeeded traumatisms, and infections, marked by the formation of foci of pus. T. J. Weller reports three cases apparently arising from chronic cystitis. Cases occurring in the puerperium are doubtless due to such a cause.

PATHOLOGY. It was formerly considered (so stated by Landray in 1859) to be without an assignable lesion, but later investigation has uniformly revealed lesions, but not of definite location. The study of one group of seven cases led the observers to conclude that it was a peculiar manifestation of peripheral neuritis, but the latest conclusion seems to be this: it is a disease which may occur with any one of four groups of lesions. First: there may be changes in the peripheral nerves which indicate interstitial and parenchymatous neuritis. Second: there may be changes in the spinal nerve-roots only, particularly in the anterior ones. Third: there may be disseminated foci of inflammation, exudation and capillary hemorrhage in the medulla and cord, and swelling of the antero-lateral tracts, and in the anterior horns. Fourth: there may be changes in all parts of the lower motor neurons, peripheral nerves, spinal roots and ganglion-cells due to a scattered and diffuse pathological process. The cortical neurons escape. It is an acute parenchymatous degeneration of the peripheral motor neurons, of toxic or infectious origin, the simultaneous and demonstrable changes in the liver, spleen, kidneys, and lymph-glands lending color to this hypothesis. Some feel that it is a poliomyelitis of a severe type, but observers of great experience find cases that conform clinically, and post-mortem, to the type described by Landray. Buzzard isolated a microörganism which he termed Diplococcus thecalis, which he thought might in some degree be the cause of the disease. He also found changes in the chromatin of the cell bodies of the anterior horn, and the Column of Clarke, while the myelin sheaths showed fatty change. The disease has been known to begin in the medulla, and extend downward.

SYMPTOMATOLOGY. Generally there are no warning symptoms, but in one of my own cases there was a feeling of malaise, with a numbness of the lips for about twenty-four hours previous to the advent of any notable paralysis. Others have noticed tingling in other parts as a premonition. Aside from this, initial sensory changes are absent, but later there may be tenderness of the muscles on deep pressure. The first diagnostic symptom is a weakness in the lower extremities, which becomes a relatively complete paralysis in from twenty-four to forty-eight hours. The weakness and paralysis are not invariably in the lower limbs, but may be in the gluteal region or thighs. Extension of the process then ensues, and is steady and rapid. If the parts other than the lower legs are first affected, the first sign of extension will be their involvement, but, in any case, after the legs are paralyzed, the trunk is next affected, and as the paralysis approaches the thorax, the respiration becomes weakened, and may be the cause of immediate death. Buzzard points out that the toes may still be moved, even when the rest of the leg is completely paralyzed; this is unlike what one sees in the paralysis of multiple neuritis. Next, the arms become weak, and finally are as paralytic as the legs, but before they are greatly affected, the nuclei in the bulb betray the inroads of the disease, and speech, and deglutition, but most vitally the action of the heart, are involved. Death is most commonly from this latter effect, and ensues so soon as these centers are affected. There may be facial and ocular palsies in slow and mild cases. The deep and superficial reflexes are abolished as the disease reaches their centers, excepting only in those cases which are so slow in their progress that there is an interval of irritation of their centers before their ultimate destruction. Since the lesion may be a diffuse one, we may find loss in one part, and exaltation in another, but loss is the final fate of all. The sphincters are not disturbed, except in very rare instances, but, owing to motor weakness, we may find a simulated retention. While the muscles lose their tone, they do not show any actual atrophy, except in those very slow cases whose rate of progress casts doubt upon the correctness of the diagnosis. During all this paralytic process sensation is undisturbed, except that once in a while a slight dulling of perception may be found, or a little pain in the affected muscles, in the latter case when the process has been a very rapid one. Temperature is sometimes elevated, albumen found in the urine, and some enlargement of the spleen; all these symptoms are confined to the infectious cases. Electric changes are absent rulably, but will be changed in cases lasting over a week (Starr). There are no mental symptoms.

COURSE. Paralysis of the lower limbs is generally complete in one or two days (three or four hours—Prince's case), and of the whole body in three or four days. Some changes are reported with a course as long as four weeks. Remissions are possible at any stage, and later exacerbations may then occur, and result in death.

PROGNOSIS. Almost always fatal in from three to four days to a week, but a small number of cases have recovered. Spiller quotes one. The hopeful condition is one where there is a stationary condition of stasis for a few weeks, and then a slow recovery of power.

DIAGNOSIS. Cases generally present no difficulty, since no other form of paralysis presents such a combination of rapidity of onset, progression without skipping of parts, absence of sensory symptoms, sphincter disturbance, and bedsores.

Differential Diagnosis. Meningeal Hemorrhage has an even greater degree of suddenness, but it has pain in the back (sensory symptom), there are spasms of the muscles, and exaltation of the deep reflexes for a short time in the majority of cases, and finally, the sphincters are surely disturbed to some extent, and if the hemorrhage is a large one, there will be bedsores and atrophy of muscles.

Peripheral Neuritis is very like some cases of the slower kind. The sudden neuritis from alcohol or diphtheria is distinguished by the presence of sensory symptoms, in the case of alcohol, and by the primary pharyngeal, or respiratory, or ocular site of invasion of diphtheria. In all forms of neuritis the history will generally be a sufficient guide; if not, the superior rapidity of progress of Landray's paralysis, and the fact that it involves the trunk before the arms, should correct the error.

Anterior Poliomyelitis paralyzes a limb, or one side, or is paraplegic in a few hours after a sudden fever (this fever may in some cases be too transient and slight to attract attention), and in a day or two regresses, so that only a part of a limb is paralyzed. This paralysis soon becomes atrophic. The paralysis is rather wide in extent, and speedily grows less in extent, but is degenerative, while in Landray's paralysis it is limited at first. continually extends, and is not degenerative.

Transverse Myelitis affects a limited area which can be marked out by very definite sensory symptoms (root-symptoms), while Landray's paralysis has none. Transverse myelitis is degenerative, producing bedsores, and affects the sphincters, which are also points of difference.

Periodic Family Paralysis is absolutely similar, and only its short duration, and the termination in recovery could guide one to a proper diagnosis in the absence of the family history.

Treatment. The patient should be kept absolutely at rest, and the limbs should be kept warm, to keep up the most efficient circulation, either by wrappings of flannel, or the continuous warm bath. Vapor baths are very useful. There is no objection to moderate massage, but while electricity may be used, employing mild currents, it has no very great promise. If a case tends to recover, faradism should be of value in the convalescent period. There is general agreement that some method of stimulation of the spinal circulation is theoretically valuable, and for this purpose friction, the negative galvanic current, irritation by sinapisms (mustard one part and flour three parts), and even the actual cautery have been advised. In one case which recovered, the whole spine had been kept constantly irritated by a sinapism. In all cases nutrition should be abundant and stimulating, and pure stimulants should form a feature of the treatment. If deglutition is affected, the nasal tube should be used, and in cases of paralysis of the glottis, tracheotomy should be made. Injections of ergotin were used in another case which recovered (J. K. Mitchell).

Starr doubts the cogency of the theory which underlies its use, but he believes that there may be a possible advantage in an antiseptic treatment. For this he suggests the hypodermic injection of carbolic acid, or the intravenous use of formalin. Bolten (Berliner klinische Wochenschrift. Jan. 16th, 1911) draws a sharp distinction between any form of poliomyelitis and Landray's paralysis, and says that the latter is a functional disturbance of motor power only, from intoxication, and the aim is to dilute the intoxicant. He reports a cure by the abstraction of 40 c. cm. of cerebrospinal fluid, and the installation in its place of 30 c. cm. of normal salt solution. This is repeated six times in eight days, so that in all 600 c. cm. are withdrawn, and 540 c. cm. are substituted. Vining and McGregor report the favorable results of treatment with strychnine.

THERAPEUTICS. The drugs which have been recommended for use in this disease are of doubtful efficiency, since almost all of them are conspicuous by the abundance of sensory symptoms found in their pathogenesis, while this disease totally lacks them in some cases, and in none of them is there more than some slight parasthesia. The recommendation that it should be treated as a myelitis is not sound, since neither the pathological findings, nor the symptoms, constantly resemble that disease. In one case of the author's the administration of Oxalic acid in the sixth potency was followed by an amelioration of the symptoms, but cardiac failure and death followed the apparent improvement. Aconite may be of use where the paresthesias are prominent in the form of tingling numbnesses. Conium maculatum has a better basis, since poisoning by it produces a paralysis spreading from below upward, and finally causing death by failure of respiration and cardiac action, all with an unclouded sensorium. Hydrophobium; after inocculation with the serum of Pasteur some cases have died with the symptoms of Landray's Paralysis. In at least one of these cases the symptoms were so similar that post-mortem examination was necessary to correct the diagnosis. The following remedies have been advised: Alumina met., Lathyrus, Ledum, Phosphorus, Rhus tox., Secale, Gelsemium, Conium, and Cocculus.

CHAPTER VI.

DEGENERATIVE DISEASES OF THE SPINAL CORD.

System Diseases.

TABES DORSALIS.

Locomotor Ataxia—Posterior Spinal Sclerosis.

Definition. This is a chronic degenerative disease of the nervous system, affecting specifically the cells of the posterior root ganglia, resulting in a more or less complete degeneration of the sensory neuron, both centrally, and peripherally, and from such degeneration arise the characteristic ataxia, and sensory derangements. While there may be an extension of the process, any motor involvement is accessory and unessential, the sole essentials being muscular incoördination and loss of knee-jerk. It is not a paralysis, but an incoördination. It is generally, though slowly, progressive, and the patient finally becomes a hopeless invalid. Among nervous diseases it is a common one.

The Symptoms of a Typical Case. A person over forty years of age begins to suffer from double vision, from lightning pains in the extremities, and from incontinence of the bladder. He is sensible that he feels compressed as by a girdle about the body, or one of the limbs. He finds that he cannot stand steadily without the aid of vision, and that he has lost impressions of touch in his feet, and perhaps also in his hands. On examination it will be found that he has lost the deep reflexes, that he has insensitive areas upon the skin, of the mammary region or soles of the feet especially, and that his pupils will react to accommodation, but will not react to light. Lumbar puncture will reveal a moderate lymphocytosis of the cerebro-spinal fluid.

Differential Diagnosis. It is to be especially distinguished from Multiple Neuritis, General Paresis, Syphilitic Pseudo-Tabes, Spinal Syphilis, Multiple Sclerosis, and Hysteria, all of which present marked similarities, and may co-exist. It is less often similar to Hypochondria, Exophthalmic Goitre (of an aberrant form), Neurasthenia, Chronic Myelitis, Spinal Tumor, and Hereditary Ataxia. Multiple Sclerosis, Ataxic Paraplegia, Cerebellar Tumor, and Syringomyelia may at times resemble it.

Age. A disease of the fourth to the fifth decade of life. Bramwell reports that 73% begin between the ages of thirty and forty, and that while one case occurred at twenty-one, and another at sixty-six years, youth and old age are practically immune from acquired tabes.

Heredity. It is doubtful whether it is ever actually transmitted as tabes from parent to child, but a sufficient number of cases have been reported of several persons in the same family being affected to raise a suspicion of its possibility. Croner quotes two series in which three brothers were affected. In the first group syphilis had been demonstrated; in the second it was positively known as to only one of the brothers. Canonnet has collected 89 cases of juvenile tabes, all of whom are attributed to hereditary syphilis. Bertolotti reports a case of hereditary syphilis which did not develope tabes until twenty-five, and another until forty years of age.

Race. It has been believed that the negro race was practically immune to tabes, although syphilis was common; still cases have been reported, and Pritchard states that it is by no means as uncommon as supposed, but that they are prone to exhibit the amaurotic form, while the subjective symptoms of the disease are absent. Gordon reports a case which also suffered from a brachial monoplegia from poliomyelitis.

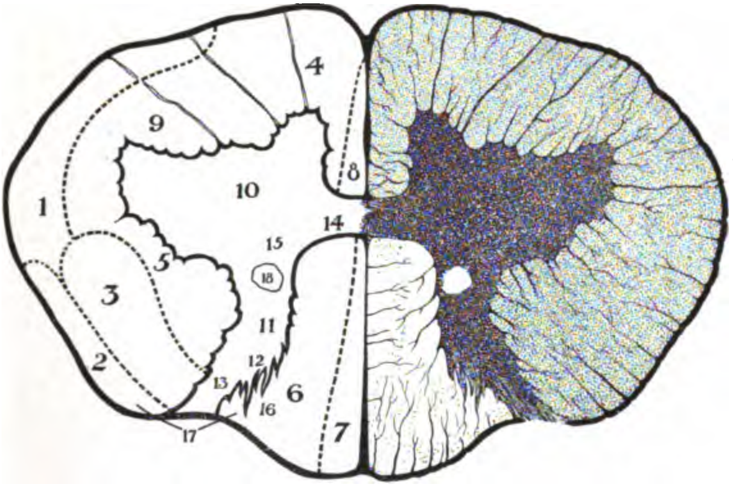


Figure 63. Tabes Dorsalis.

Site of degeneration is shown in the right half as a white area. By referring to the left (schematic) half it will be seen to have wiped out the tracts of Goll (7), and Burdach (8), and to have affected the fibres and secondarily the cells, to some extent, of Clarke's column (18).

Etiology. Since the discovery of the spirochaeta pallida in the spinal cord and brain of tabetics, syphilis has been acknowledged to be the basis of the disease. There seems to have occurred in the course of time some change in the behavior of the organism, since the changes in the nervous system are not like those produced by the spirochaeta in the earlier stages of its activity. Therefore tabes is still termed a parasyphilitic disease The present conclusions stamp with approval the original proposition of Gowers that "Syphilis is a 'conditio sine qua non' of the occurrence of tabes." Since only a small per cent. (2 to 5) of syphilitics develope tabes, it has always been believed that it was syphilis plus *x* (x being an unknown and intensifying element). Edinger considers that, among others, tabes is more of an exhaustion disease than a toxic one; that is, there is a poison plus a functional over-use of a part, which combination produces a using-up disease, or *Aufbrauchkrankheit.* It has been said with some show of plausibility that the parasyphilitic will be a tabetic if a mechanical worker, and a paretic if a brain worker. That, if true, would indicate that exhaustion was not only a deciding, but also a localizing factor. Traumatism, fright, over-exertion, and refrigeration have now been relegated to their proper place, i. e., exciting causes only. There are sound clinical grounds for the belief that these conditions are competent to produce this result.

PATHOLOGY. Gowers has recently stated that the symptoms of tabes are produced by the action of the chemical toxin derived from the syphilitic poison. This toxin acts as a ferment on the albuminous bodies, which renders them a poison instead of a food to nervous tissues. The lower sensory neuron is especially susceptible to this toxin.

Von Pirquet formulated the conception of "Allergie," which was the change in the character of the reaction of tissues to poisons after infection. If it was increased, and they responded more and more readily to the poison, the condition was called "Hyperallergie:" on the other hand it might go on to complete lack of response, which is called "Anergie." The Hyperallergie is the same as the Anaphylaxis of Richet. Acting upon this suggestion, McIntosh, Fildes, Head, and Fearnsides have concluded that parasyphilitic phenomena are manifestations of hypersensitiveness, and this being the case the tissues would react to such slight doses of the poison that it is not peculiar that we have difficulty in finding the spirochaetae in gumma and parasyphlitic tissues. They regard parasyphilis as a purely clinical conception; it is a diseased state which may affect any portion of the brain and the spinal cord; that tabes and paresis can only be erected into two clinical divisions by an arbitrary selection of signs and symptoms; that the difference between the gumma and purely nervous lesions depends only upon the nature of the tissues attacked. In the first it is the reaction of hypersensitive vessels and connective tissue, structures which show a proliferative change, and where proliferative repair may occur at any time. In the second case it is the response of highly sensitized, highly differentiated nerve elements which have no power of regeneration. Up to a very recent period it could pertinently be asked, "Why is the central nervous system so especially affected by the later activity of the syphilitic virus, and why is the sensory neuron so selectively attacked?" Since the spirochaeta pallida has been demonstrated lying deep in the substance of the brain and spinal cord, the first part of the question needs no further answer, while the relative vulnerability of the sensory neuron is explained thus: the cerebrospinal fluid is the bearer of the infection, and just as the sensory nerve-fibres enter the spinal-cord they are bared of their protective envelopes, including the sheath of Schwann. This structure has both a protective and a nutritive role. A reactive process which is sufficient to destroy a nerve-fibre not provided with this sheath may be insufficient to injure a fibre protected by the neurilemma, while on the other hand this structure is an essential factor in any process of regeneration. This is assumed to be the reason why the sensory, and not the motor fibres are affected, and may be the reasonable explanation of the localization and frequency of "Crises," which are actually attacks of meningo-radiculitis.

Finally, the incidence of the infection of tabes is to be reflected on the cells of the posterior root ganglia, possibly due to that form of cell degeneration termed "reaction at a distance," but ultimately resulting in degeneration to a variable degree of the centripetal division of the bifurcated axis cylinder of the cell, and less obtrusively of the centrifugal branch. The resulting degeneration of the centripetal fibres produces areas of sclerosis in the posterior columns, so long considered the initial lesions of the disease. In the early stages of the disease, or in cases of a moderate degree of severity, the degeneration is confined to a crescent-shaped area lying to the inside of each posterior horn, since the posterior median area contains, beside the exogenous fibres coming from without, a crescent-shaped tract on the margin, and one near the anterior border of the posterior median fissure, which are associative and endogenous, i. e., they originate in the cells of the posterior horn, and not in those of the posterior root ganglion. As the duration and severity of the case increases, these tracts also succumb.

SYMPTOMATOLOGY. As a general truth it may be said that from ten to fifteen years after an infection from syphilis (Bramwell says 45% in ten years, and only 10% after twenty years) the patient will find himself suffering from certain disabilities, for which he will seek advice from a physician. Among possible initial symptoms there is a wide variability as to which will first attract his attention, and investigation has frequently revealed that certain deviations from health had long existed, which he had only casually noticed, and had endured without having investigated their meaning, or which had been so transient that they had passed out of his mind. Lightning pains are the first symptom in 51%; diplopia in 7%; dimness of vision in 7%; ataxia 6% (Bramwell); bladder symptoms first in 30% (Erb). As a rule, the patient is most liable to complain first of darting pains in the legs, or that he cannot hold his urine, or that he has a great deal of vertigo, and, once in a while, that he has become so ataxic that he cannot go about a room in the dark, or maintain his position while he bends over the bowl to wash his face in the morning. A patient of my own first discovered that he was ataxic from getting into trouble frequently, on account of bumping into people upon the street. Sometimes a recurring causeless abdominal colic will attract attention, or it may be the presence of double vision. With these pains, or with most of the other symptoms mentioned, he may have little conscious ataxia, and so he should be placed in the Pre-ataxic stage, which may last from a few months to ten or more years. Even at this time, however, a careful examination will disclose a diminished, and probably a lost knee-jerk, an Argyll-Robertson pupil, and in some parts of the body there will be an inability to correctly appreciate touch and pain, especially in the pectoral region, or in the soles of the feet. If it is found that he will stagger if induced to stand erect with the eyes closed, and the feet closely apposed (Romberg's Sign), he has already entered upon the second, or Ataxic stage. Sexual power may be diminished, and perhaps lost. It will now be found that he cannot execute movements accurately with the eyes closed, nor can he walk well without the aid of sight. By this time there are very definite losses to all sorts of sensations of the skin, his bladder and rectum are under poor control, he is tormented by pains in the limbs, and pressure symptoms about the body, and his vision is probably much impaired. This stage may last from two to ten years, with long halts, and some remissions to his suffering, but soon there will be an increasing weakness, the appearance of atrophies about the joints, and when they have reached their full developement he has entered upon the third, or so-called Paralytic stage. This is hardly ever a true paralysis, since he generally has the power to execute any required movement (unless the joint disintegration has reached a high grade), but his incoordination is so great that his power is misdirected. His mind now tends to degenerate in the direction of a mental feebleness, rather than into any actual or definite psychosis. In order to more specifically describe these changes, they will be taken up as they are classed in the following table (from Church and Peterson), in the order of their rulable sequence:

1st. Pre-ataxic stage. 6 mos. to 20 yrs.	2nd. Ataxic Stage. 2 to 10 yrs.	3rd. Paralytic stage. 2 to 10 yrs.
Motor. Eye-palsies.	Less.	
Ataxia.	Increased.	Increased.
Muscular weakness.	Paresis.	Paraplegia.
Sensory. Pains.	Pains.	Pains less.
	Anesthesia.	Increased.
Reflexes. Knee-jerk lost.	do.	do.
Argyll-Robertson pupil.	do.	do.
Trophic. Arthropathies.	More rare.	Rare.
Visceral disturbance.		
Sexual weakness.	Increased.	Increased.
Vesical weakness.	do.	do.
Constipation.	do.	do.
Special Senses.		
Diplopia.	Rare.	
Optic atrophy.	do.	Increased.
	Deafness.	do.
		Paralysis of accommodation.

Eye Palsies. All the muscles of the eye may be paralyzed, but the ciliary and trochlear almost always escape. Usually we find ptosis (drooping of the upper lid), and squint (strabismus) usually fleeting at first, lasting from a few days to a week, but finally becoming permanent, and the squint produces diplopia (double vision). It may not be apparent to the patient, and be first discovered on examination of the eye for other losses. A ready test is made by covering one eye, warning the patient to keep both open, and having the patient follow with the uncovered eye the movements of the examiner's finger. This should be moved in all directions, finally ending in causing extreme outward rotation of the eye. Now quickly uncover the other eye, and observe the relative position of the two eyes. They should preserve their parallelism, in spite of the fact that one has been covered. Examine next, ending in extreme inward rotation. It is diagnostic that it is from tabes when it is not relieved by specific medication, for usually the only other probable cause is syphilis.

Ataxia. Under the above heading we should class several defects, which are often viewed as separate conditions, thereby obscuring their common origin, namely, Hypotonia, Incoördination, Static Ataxia, and Asteriognosis. These are manifestations of a common loss, namely, the lack of appreciation of pressure and tension on skin, tendons, muscles, and joint surfaces.

Hypotonia. This is the loss of the power of smoothly executing movements with the weighty parts of the body. The patient betrays it by dropping into a chair, instead of gradually seating himself; or, more accurately, if the patient, lying on his back with the knees bent, and touching one another, and with covered eyes, is asked to permit the legs to smoothly and slowly fall apart. He cannot accurately estimate the relations between muscles and their antagonists, and therefore executes the movement in a jerky, hitchy manner. It is said by some to be absolutely the initial symptom.

Incoördination. This is essentially a sudden and immoderate action of the prime movers of the body, on account of a lack of appreciation of the proper amount of resistance in the antagonistic muscles, since no movement can be smoothly executed without a nice adjustment of the two sets of muscles. A simple test for this loss consists in the examiner touching the heel of one leg upon the patella of the other. The patient is then asked to repeat the maneuvre, with closed eyes. He will then grope about with the heel, thus making manifest the degree of incoördination. If the tabes affects also the upper portion of the spine, the patient may be asked, with closed eyes, to touch the tip of the nose with the tip of the first finger, the arm having previously been extended to its full length out at the side of the body. The handwriting will also show this same jerky incoördinate movement. Such movements can be very accurately executed by a person in health without the aid of vision.

Static Ataxia. This is at times the first symptom, but may exist undetected for a long period, since it is not painful, nor very obtrusive. It consists in an inability to walk or stand steadily without the aid of vision. It is revealed by Romberg's test (described above), and depends upon the presence of anesthesia of the soles of the feet (a similar loss occurs, at times, in the hands), and the loss of appreciation of tension in muscles, joints, and tendons. It naturally is an early result of these losses, since both standing and walking depend upon a constant and delicate readjustment of muscular effort, and the tabetic has lost many of the nervous end-organs and tracts which formerly furnished him with this information. Closing of the eyes immediately increases greatly ataxia of both station and gait. Static ataxia rulably increases as the disease progresses, but (with scanty evidence) it has been asserted that as sight fails, static ataxia disappears.

Lymphocytosis occurs in tabes from the constant meningitis.

Asteriognosis. In a state of health, we can, within 5%, appreciate the difference in weight of two similar objects held in the hands, without the corrective aid of vision. More than this, we can appreciate slight differences of form and texture, or peculiarities of a surface. The loss of this faculty is not a constant symptom in tabes, and it occurs also in other diseases, since it is perfect only when the receptive centers of the cortex, the sensory conduction paths of the cord, and the receptive peripheral apparatus are in a healthy condition. It depends upon the perfection of the spacing sense, the localizing sense, the muscular sense, the pressure sense, the temperature sense, and the pain sense. This is Steriognosis, and its absence is Asteriognosis.

Muscular Weakness. This is generally an early symptom, for there is always a degree of anemia, and it endures throughout the disease, but in only 5% is there any general paralysis, and whenever it occurs, it is a complicating condition. Separate muscles exhibit normal strength, even in the late stages of the so-called paralytic phase. The helpless invalid will execute simple movements with adequate force, his actual loss being that of incoördination. Of course in the presence of extensive joint-destruction he will necessarily be helpless. The accessory paralyses indicate that the nuclei of innervation, or motor tracts, have been invaded, but such cases are rare, and the results are generally temporary. The most common paralysis is ocular, then facial, next lingual, and finally laryngeal. We may find paralysis of a single part (monoplegia), or of the lower limbs (paraplegia), or of half of the body (hemiplegia), and often the patient suffers from a sudden giving-way of the legs, which is a transient form of paraplegia.

Sensory Disturbances. Since tabes is essentially a disease of the sensory nervous system, we should expect to find all varieties of change in sense-perception. These changes may be subjective, like the paresthesias, numbnesses, creepings, and crawlings which are such an early and constant symptom. Actual loss, of which the patient may for a time be ignorant, is usual, and Oppenheim says that the pectoral region, and the inner aspect of the legs may be initially the only sign of tabes. This loss, together with anesthesia of the palms, and soles, and face at times, is most diagnostic when associated with other symptoms. The loss in the tips of the fingers is often first discovered when the patient finds that he cannot feel well enough to button his clothes. In almost all cases anesthetic areas will be found in strips or spots irregularly distributed over the body. In some cases it is possible to pick up the skin in the fingers, and move it about without the patient being able to correctly state the direction of the movement. This loss may be for all varieties of sensory stimulation, touch, pain, heat, or cold. Racine says that Abidie's symptom is present in 80%, i. e., the absence of pain on strong compression of the tendo Achilles. Biernachi's sign, analgesia of the ulnar nerve, is more frequent than analgesia of the skin of the inside of the arm, and if bilateral, is very diagnostic with the other. The testicles may also be analgesic when compressed. Thiess reports the painless confinement of a tabetic woman. The paradoxical reflex is also present at times, i. e., pressure is interpreted as pain, or the contrary, or pain inflicted upon one part is felt in another, or perception is correct, but delayed. These sensory losses, both objective and subjective, are always present in the second stage, and are not confined to the surface. Jean Heitz asserts that the loss of bone sensation antedates the skin losses. He applies to the bony surface a tuning-fork giving 128 vibrations to the second, and while these vibrations are perceptible throughout the bone in health, they are lost in tabes.

Pain. In all but a very small proportion of cases (9 out of 450 in Starr's group), pain is a symptom, and is apt to be an early one. If early, it commonly continues throughout the disease, but may disappear with the appearance of marked ataxia. If pain appears late in the disease it is apt to be mild, and of short duration. At first it is often assumed to be rheumatic, or neuralgic. Pains are of two varieties: brief, and prolonged. The brief pains are of two classes: superficial, and deep; the brief superficial pains are usually felt on, or just under the surface, and usually in one spot. They are extremely brief, but recurrent, and from their character have gained the name of lightning pains. They have the remarkable effect of leaving the skin *very* tender (*Plaques douloureux*), and may occur where the skin has lost its sensibility to pain from external irritants. They most commonly occur in the limbs, especially the lower legs and feet. Next are brief, deep-seated pains; not as a rule very definitely located, but sometimes referred to the joints, usually in the limbs, and not followed by hyperesthesia. They are not so momentary as the superficial pains, but last several seconds. Prolonged pains: these last for days or hours in the same place, most commonly in the trunk, and are usually deep-seated, simulating sciatic neuritis, the only superficial variety being the so-called girdle-pains, which are generally about the waist. These pains consist in a sense of distressing tension in muscles and tendons, but they may have a burning character. There is seldom any exciting cause, since they are the logical results of pathological changes in nervous structures. Formerly it was considered that they arose from changes in the peripheral end-organs of the sensory nerve, but they are now recognized as root-inflammations (meningo-radiculitis) manifested in the area of distribution of these nerves. Root-pains are most usual, says Starr, in the 2nd and 3rd lumbar nerves, and therefore the pains are felt initially in the front of the thighs, and tend to extend downward at first, and later upward. A patient of my own has most typical root-pains in the intercostals.

There is a form of the disease where pains so dominate the scene as to justify a certain classification. Gowers reports 11 such cases, all in adult males, and with a specific history in 9 out of the 11. The pains were severe, and neuralgic in character. Girdle-pains are like a burning band around the abdomen, or sometimes like a narrower band about the limbs. Another form of this nervous reflex is a sense of pressure, hardly a pain, and of much broader distribution about the upper abdomen, or the chest ("tabetic cuirass"). From the same causes arise spasmodic pains in the viscera. which may exist in many situations, and are termed "crises." A crisis is a subacute meningo-radiculitis. The gastric and visceral are the most common, and appear as acute gastralgias and intestinal colics without adequate cause. There is a more chronic form ,which is like a chorea of the stomach with a spasm of the inlet and the outlet, due to the untrammelled action of the vagus when it is released from inhibition by the degeneration of the sympathetic (Loeper and Oppenheim). Eichhorst says there are three kinds of gastric crisis. A neuralgic form, not accompanied by vomiting, but increased by light pressure, and at times relieved by deep pressure. A hypomotor form which leads to vomiting, with a variable vomitus. A variation is a tetanic form (the Choreic form of Loeper and Oppenheim), where the stomach can be mapped out by palpation, and there is severe pain, which ceases when the spasm relaxes. Lastly there is a purely secretory form of gastric crisis, with the vomiting of an acid vomitus, but containing little or no hydrochloric acid. Loeper remarks that there are three types of intestinal crisis. A simple form like lead colic, which may last for as long as twelve days, with absolute constipation, and retention of urine, but no fever. Next the Choleroid form, with violent cramps and diarrhea. Lastly the enteric type, where the patient exhibits great tenderness of the whole abdominal wall. There are testicular crises, those of the clitoris, nephritic, hepatic, and Oppenheim speaks of those of the larynx: this latter is a painful sort of croup attack, coming on in an adult, and sometimes threatening suffocation. It may be precipitated by pressure on the hyoid bone, and therefore a tight collar should be avoided by those suffering from this form of crisis. Harlan says that laryngeal crises are very rare. Cardiac crises simulating angina pectoris have been described, but must be accepted with extreme caution, and only after prolonged observation.

Tabetic ocular crises have been reported by several authors, and Spiller, Jour. American Med. As'n, Mar. 18th, 1916, reports a case with intense jerking, stinging, burning pain, the eyeballs feeling as though they would come out of the orbit, accompanied by free secretion of tears. They may suddenly remit, and begin again with great severity, and at times the patient has visual auræ of various colors, and objects.

Reflexes. The deep reflexes are always affected in tabes, and speedily lost, without the previous exaggeration found in some other diseases where loss is also the final result. The typical symptom of tabes is absence of knee-jerk, but the Achilles-jerk is said to be an earlier loss even than the knee-jerk, and the triceps-jerks are also lost. The value as a symptom of the discovery of lost knee-jerk (Westpfal's sign) rests upon the fact that most observers affirm that it is never absent in health. Some believe it to be absent in 1-2%. It is never lost in any condition except transiently, unless organic disease is present. It may be a persistent loss in diabetes. It may be temporarily lost in some febrile, and post-febrile conditions, for a time after an epileptic attack, and in acute alcoholism. It should not be considered lost unless the examination has been a very thorough one, since anxiety may mask it, or it may be restrained. The method of examination is as follows (see Fig. 10): have the patient cross the leg over its fellow at the knee, and, being sure of the position of the patellar tendon, strike it a sharp blow with the edge of the hand, or a percussion-hammer. If the patient is in bed, support the leg under the lower part of the thigh, and proceed as before. If there is no response, place the patient in a sitting posture on the edge of the bed, or better a table, with the legs hanging just free of the edge, and repeat the blow upon the tendon. Failing in these ways, do not consider that its absence is certainly established. Now use some method of reenforcement, all of which are based upon the idea of attracting the patient's attention from the matter in hand. The most common is that of Jendrassik (see Fig. 11). Place the patient as before, then ask him to hook his hands together, and fix his eyes upon the ceiling. At the word of command he is to attempt to jerk the hands apart by an instantaneous pull. At the moment of command the examiner strikes the tendon as before. If no jerk can be elicited, the reflex may be considered to be absent, but if possible the maneuvre should be repeated at a subsequent date, lest there may have been some error in technique. Another method has been recommended. Give the patient a somewhat heavy book, and ask him to read aloud. Under the combined mental and physical strain he is unable to inhibit the reflex, and it is said to be applicable to children who can only read the letters. Exceptionally the knee-jerk may persist in tabes; viz., if the lesion in the cord is confined to a very definite and small area, which is either too high, or too low, to involve the reflex arc. Bramwell reports a case where the plantar reflex was exaggerated, and the knee-jerk present; and one where the Achilles-jerk was absent (see Fig. 12), and the knee-jerk persisted.

Superficial or Skin Reflexes. These are not constant in their condition, being exaggerated in some cases; but even if exaggerated at the outset, they are usually diminished or lost in the later stages. In one case an exaggeration of the abdominal reflex antedated all other symptoms by two years, and an exaggeration of the abdominal with loss of the knee-jerk is said to be an early symptom of the disease. The cremasteric reflex (sharp retraction of the testicle when the skin of the inside of the thigh is stroked) is diminished or lost. The center for this reflex lies in the third lumbar segment. Its absence on both sides is significant, and means very probably an organic nervous disorder in those cases where testicular disease, excessive masturbation, etc., can be excluded. Absence on one side is of less importance, but has the same meaning. The reflex has about the same significance as the umbilical. The reflexogenous zone is frequently very extensive; in about 30% of the cases the reflex was elicited on plantar stimulation. Tonicity of the sphincter ani is diminished or lost.

Organic Reflexes. A disturbance of the functions of the rectum and bladder is an early symptom in 80% of the cases. Erb says disturbance of the bladder function is an initial symptom in 30%, and arises from the imperfection of the sensory apparatus. The common rectal difficulty is an obstinate constipation, but once in a while, involuntary defecation will harrass the patient. The bladder fails in function by a loss of the control of the sphincter, or, even earlier, from an anesthesia of the urethra; the patient may wet himself, from an inability to appreciate when he has finished micturition. Instead of relaxation there may be retention, or slowness in starting the stream, but catheterization should be deferred, since after some delay, or in some position, the patient is usually able to perform the act. These defects become more pronounced in the later stages of the disease.

Sexual Power is lost in 50% of the cases, and is said never to return, but the author saw one example of a complete return of sexual power; it remained competent for a year or more, and then the patient passed out of his knowledge. This is one of the losses which leads a person to consult a physician. It also occurs in diabetes. If the cervical cord is invaded by the disease, it may cause a more or less constant priapism. If this priapism precedes the loss of sexual power, or follows it as a later symptom, sexual gratification will increase the pains and the ataxia.

Pupillary Changes. These are actually organic reflexes. These changes are of great diagnostic importance in tabes. It is very significant, and should stimulate one to further investigation, if the pupil is found to be of unequal size in the two eyes, or to differ from the normal by peculiarities in shape or position. The most distinctive pupillary condition in tabes is the Argyll-Robertson pupil. The pupil will react normally to accommodation for objects at variable distances, but has lost its power to modify its aperture proportionately to differences in the amount of light to which it is exposed. The reaction to accommodation is better preserved than that to light, since the duration of the stimulus is so much longer that the blunted perceptive power can receive the impress of the one, and fails in the other. Do not feel that the condition is proven if the reaction to light is absent, for the test of accommodation is needed to determine that the eye has not undergone previous change which would neutralize the test. This is especially true in elderly persons, in whom accommodation is very greatly reduced. Test for accommodation by observing the dilatation of the pupil when the eye is fixed upon an object at a distance of ten to twenty feet, and its subsequent contraction when the gaze is fixed upon the examiner's finger, approached within eight to ten inches of the patient's eyes. The difference in the width of the pupil should be marked. Examine the light reflex thus: face the patient toward a good source of light, and then observe if the position is such that the outline of the pupil can be clearly seen. Some care must be used, since some eyes will reflect confusing images unless the illumination is somewhat lateral. This is peculiarly true of the brown and black eye. Now cover both eyes with the examiner's hands, warning the patient to keep both of them wide open, and mentally focussed upon some distant object. On uncovering one eye quickly, the pupil should contract sharply. Slowness of response is evidence that it is only a stage of a process that will end in loss. Try both eyes, since the loss at first is occasionally unilateral. If the patient is in bed flash a match, light from a candle, or other source of illumination before the eye. Holding the lids closed, and suddenly pushing them up may be sufficient. The investigation may require considerable care, as the pupil of the tabetic is apt to be small, as well as stiff.

While this is an early symptom of tabes (90%), it is not pathognomonic. It occurs in primary optic atrophy, primary lateral sclerosis, amyotrophic lateral sclerosis, in general paresis, in progressive muscular atrophy, at times in constitutional syphilis, in poisoning by bisulphide of carbon. It has been noted as a temporary condition after railroad accidents, and other shocks, followed by no evidences of a grave neurosis, and exceptionally in alcoholism. That is, it may be a temporary toxic symptom (Kiernan). Its pathology is doubtful, although it probably depends upon a degeneration of the ciliary ganglion. Fuchs says that there is Argyll-Robertson pupil, then spinal miosis, and finally anisocoria (irregularity in the diameter of the pupil). Total rigidity, or enlargement of the pupil, with paralysis of accommodation speaks for syphilis. Schillenhelm says that Argyll-Robertson pupil is a more useful symptom in the early determination of tabes than the loss of the knee-jerk.

Optic Atrophy. This may come on at any stage. From 1 to 2% have it as an initial symptom, and from 20 to 35% have it in some stage of the disease. Fisher and Fuchs say that optic atrophy is rare in paresis, and common in tabes; Dana says that it is present in the pre-ataxic stage. It may antedate all other symptoms by years; it is of the gray variety, generally bilateral, and occasionally it is preceded by choked disc. It never coexists with strabismus, or ptosis. The patient may be unconscious of his loss for years, since central vision is likely to be preserved for a long time. The loss may be in the width of the field of vision, a section of the field may be cut out, or there may be no blindness, but only a dimming of the whole field. There may be a loss of one-half of the field (hemianopsia) and the loss may be bitemporal, such as we find in multiple sclerosis, except that the atrophy may be gray instead of white. At times there is a loss of perception of one or another color. It has been asserted that as blindness comes on ataxia diminishes, but the author's experience proves that this is, to say the least, not universally true. None of the special senses are so often affected as that of sight.

Deafness. Vertigo is a common symptom, and it is sometimes associated with hardness of hearing, which is at first unilateral, and only to the high notes, but finally is on both sides, and to notes of all sorts of pitch. It is seen in about 1%, and in the absence of an hereditary tendency, its presence should suggest the possibility of tabes.

Atrophies and Arthropathies. Whether or not there are definite trophic centers in the cord, atrophies always follow a general deterioration of the peripheral sensory apparatus. Gowers says that he has never seen a case of atrophy without a preceding cutaneous anesthesia. These atrophies affect not only the superficial, but also the deeper tissues, just as do the changes of sensation. Superficially the skin may show glossy skin, eruptions like pemphigus, and ichthyosis, etc., and there may be subcutaneous ecchymoses, or widespread effusions under the skin, and herpes zoster is often seen. A case is reported in a male, forty-eight years of age, who had a sudden attack of edema of the face, especially of the upper lip, and upper lids. A still deeper condition is that of perforating ulcer, which condition is almost confined to tabes. This occurs mostly in the soles of the feet, but may occasionally affect the hands instead. It may occur in the mouth; first the teeth loosen, then the alveolar margin of the jaw is absorbed, and if it be in the upper jaw, there may be perforation into the nasal cavity. The nails may fall off, and the teeth drop out, all without pain. The deeper effects of the atrophic condition are shown in the fragility of the bones, in which fractures occur so easily that they have been spoken of as spontaneous. Painless fracture should always make us suspect the presence of tabes. Taylor concludes that spontaneous fractures are not infrequently an initial or early symptom of tabes. Frequently such fractures occur before there is any disturbance of gait. All adults who have painless or spontaneous fractures should be examined for tabes. Tabetics should avoid sudden or jerky movements, especially heavy lifting, or twisting movements. The most characteristic of all atrophic conditions is that where the articular surfaces of the joints are disintegrated. This is most common in the knee-joint, and in patients of the female sex. The ankle is the joint affected next in frequency, and the wrist much less often, but the joints of the fingers and toes are exempt. This condition is known as the "Charcot Joint," and its symptoms are the sudden onset of an enormous whitish swelling of the surrounding parts, and absence of pain, disintegration of the joint, and loss of function. It may be an initial or early symptom of tabes. Gowers says that the dystrophies of tabes are the result of the degeneration of the sensory nervous system, as no specific trophic nerves exist. The deep or sensory nerves are affected equally with the superficial, and therefore two results follow. 1st. The condition of the tissues is not appreciated by the patient, and 2nd, the nerves do not functionate to rebuild or build the structures of the body. He believes that bedsores and arthropathies are not essentially tabetic in origin, but may be the untreated results of traumatisms, or intoxications like rheumatism. He feels, therefore, that if we would more carefully examine our cases, we could properly treat such cases in their initial stage, and then there would be no arthropathies. Persistent attacks of shooting pains in the trunk or limbs should always lead to an examination for tabes. The term tabes dorsalis should be preferred to the misleading "locomotor ataxia," since an ataxic gait is not infrequently one of the latest symptoms to appear.

Heart Lesions. Nearly all cases of advanced tabes show valvular lesions, especially prone to affect the aortic orifice, but due probably to the initial syphilis, rather than to the present tabes.

Mental Symptoms. These may occasionally be present, not as a distinct psychosis as a rule, but only as a condition of relative apathy. The mentality of the tabetic is usually so clear that he can transact business with propriety, and often with success. There are cases where the cerebral endarteritis, which is a heritage from the original syphilis, produces definite mental changes. These are not always permanent. The case of a woman is quoted who was the victim of expansive delusions, having fainting fits, but no convulsions. A month later she revealed all the classical signs of tabes, and three and a half months later she regained all her mental acuteness.

Rare Symptoms. Lingual hemiatrophy, abductor palsy of the larynx giving rise to difficulty of respiration, rupture of the tendo-Achillis, and muscular atrophies in almost any part of the body. The disease is occasionally complicated by exophthalmic goitre, paralysis agitans, neurasthenia, hysteria, or diabetes. Hoover says that apnea may be a symptom, and occur after a small dose of morphine, or automatically. Eshner reports a case of tabes and paralysis agitans in the same patient. Tooth and Howell report a case where the motor neuron was also implicated. Occasionally the picture is one of progressive muscular atrophy. Hunt reports that out of 164 cases at Cornell clinic there was paralysis in 16. At autopsy, cortical lesions similar to those of General Paralysis of the Insane (paresis) were found in tabetic cases who in life had shown on mental changes. Pregnancy is not liable to be disturbed by tabes, except that pain will be absent.

PROGNOSIS. Cure is in the highest degree improbable, and has been said by many to be impossible, but the disease in itself does not directly tend to death, though their anemic condition, and imperfection of function, continually expose such persons to danger. The shortest fatal case reported is one year, and the longest twenty-five years; the average duration of fatal cases was eight years and two months. Cases may generally be expected to reach a condition of permanent invalidism in about ten years, but the grade of invalidism is a very variable condition. Babinski reports a case of a female tabetic which existed in a benign way for forty years. The basis for prognosis rests upon the clinical type of the disease, the anatomical type, region of the cord involved, sex, age, occupation of the individual, and his treatment. Pritchard says that the hypotonic cases are speedily fatal. The halts between the definite stages, or between the evolution of definite symptoms, may be years in length, and during these remissions reeducation of the muscles may make the patient relatively better than he had been before the treatment was inaugurated. There is a small group of cases which may live from ten to thirty years without any manifestation of the disease, except continued attacks of pain. Others show a progressiveness, with changes in the optic nerve, but with little ataxia, or other tabetic symptoms. There is another group where the muscles seem too long and too lax, and allied or combined with it are arthropathies. Then there is a small group where there is an involvement of the motor system, with atrophies of the lower limbs, and with weakened muscles. In the vast majority of cases, ataxia is the prominent symptom, and re-education may neutralize many of the effects of the disease. Those cases with a marked degree of muscular insensibility, and those with a very definite hypotonia, have a bad prognosis. Some cases have been reported cured, and the cure and previous condition have been vouched for by the most competent observers.

DIAGNOSIS. The diagnosis should be by lumbar puncture if possible, as that will at once reduce it to a distinction between Tabes and General Paresis, the treatment of which is the same, if intra-spinal salvarsan therapy is employed. In both these diseases the spinal fluid is normal, except for an increase in large lymphocytes. In the absence of this test the diagnosis rests upon the discovery of a loss of knee-jerk, static ataxia, Argyll-Robertson pupil, lancinating pains, loss of sexual power, girdle-sensations, crises, changes of sensation, and hypotonia. Not all of these are present in tabes in even the majority of cases, so that we may say that if a case presents lost knee-jerk, and static ataxia it is probably tabes, and that some of the symptoms detailed in the symptomatology will be found to be associated with these, and make the diagnosis certain.

DIFFERENTIAL DIAGNOSIS. Neurasthenia simulates tabes very closely at times, and may be associated with it. A diagnostic difference is the variable character of the symptoms in neurasthenia, and their permanence in tabes. Knee-jerk may apparently be absent in neurasthenia, but care will elicit it, and it may be found at another examination to be exaggerated. An apparent Romberg may be present, but its cause is weakness, and not ataxia, and it will not be evident after rest. Fatigue is also very easily produced in neurasthenia. Apparent crises of the bladder are seen in neurasthenia, but they are due to the irritation of the bladder from the abundant salts from tissue waste, or from neuralgia. Argyll-Robertson pupil, gastric crises, and analgesia are never found, but girdle-pains are seen at times.

Hysteria has many points of similarity, and here the order and sequence of symptoms is often the best guide. There is never loss of knee-jerk, nor is there an Argyll-Robertson pupil, nor optic atrophy.

Chronic Myelitis. This is a true paralysis, and not an incoördination. Atrophy is immediate, and pronounced, and is not of a mild type, and general character, but is localized and extreme in those parts innervated by definite segments of the cord. There is pain in the back in a definite area, and the knee-jerk is exaggerated, unless the myelitis is in the segments making up the reflex arc, when it is lost. There is no disturbance of the special senses, but the sphincters are incontinent.

Hereditary Ataxia developes in the period from puberty to the twentieth year, while tabes is from the fortieth to the fiftieth years, except in the rare cases where hereditary syphilis is the cause. Friedreich's type has a loss of knee-jerk (in Marie's it is increased), and the patient has a great deal of static ataxia, but no Argyll-Robertson pupil, or other eye-symptom, except almost certain nystagmus (oscillation of the eyeball in certain positions), which is the one eye-symptom that the tabetic *never* suffers from. He has an apathetic mental state, somewhat like that of tabes, but he never has the lightning-pains, or bladder symptoms of tabes, but he does have contractures, talipes, and claw-hand, which we do *not* find in tabes. There is no pes cavus. nor lateral curvature of the spine in tabes.

Multiple Neuritis. This may complicate, and it then makes the case contradictory in its symptoms. There are also cases with much ataxia, and little palsy, which are also difficult, the pseudo-tabes cases spoken of later. Pure cases however strike us at once as a palsy, and not an incoördination. A history of some toxemia, or of some recent infection, can generally be obtained, and the onset is rapid, while that of tabes is conspicuously slow. The bladder and rectum are rarely, but certainly not permanently affected in multiple neuritis, but such disturbances are early and usual symptoms in tabes. Pains of a lightning character, crises, and cuirass-sensations are absent, and if there is anesthesia, it is of a glove and stocking-type, avoiding the trunk and face, and not widely distributed in strips and patches as in tabes. If the Argyll-Robertson pupil is present, it stamps the case as something other than multiple neuritis, but if the pupil will react *neither to light nor to accommodation,* the diagnosis is doubtful, until the lapse of time shows that it is not multiple neuritis. There may be root-pains in multiple neuritis, and there may be ocular disturbances. Early muscular wasting, and the reaction of degeneration are not tabetic, nor are the nerves swollen and tender to touch in that disease.

Syphilis of the Spine, Syphilitic Pseudo-Tabes, and Subacute Syphilitic Meningitis. These are conditions which are more nearly allied to tabes than any other, since the patient may actually have lightning-pains, paresthesias, and ataxia, and the knee-jerk will apparently be lost. It is to be elicited if the examination is a careful one, although it may be greatly reduced. There are hyperesthesias of the trunk, and pain in the back, but these are distinctly worse in the night, which is a syphilitic, and not a tabetic characteristic, and the Argyll-Robertson pupil, and bladder and rectal symptoms are wanting. There is a diagnostic difference in the fact that syphilitic symptoms are rapid in onset, they differ widely in distribution from day to day, or on the opposite sides of the body, and they may be totally absent for a time, and then return again. There are actual palsies also, and these indicate an implication of the lateral columns, or motor nuclei), a paralysis of the iris on one side, or its contraction, indicating involvement of the nuclei of the sympathetic (probably). In all such cases the case is not pure tabes; it is some other disease, or some complicating condition, since oculomotor palsy of a transient type is the only paralysis commonly attending tabes.

Other forms of pseudo-tabes are types of multiple neuritis which are caused by diabetes, the metallic poisons, and sometimes by alcohol. The diabetic form is like tabes in loss of the knee-jerk, and an ataxia, which is, however, neurasthenic in type. Sexual power is sometimes lost. The actual differentiation rests upon the presence of glycosuria in diabetes alone, and in the absence of the Argyll-Robertson pupil in diabetes, and its presence in tabes. Cases arising from the intoxicants also lack this last symptom, and on the whole are not as confusing.

Multiple Sclerosis. In typical form it is not similar, since, while the gait is ataxic, the knee-jerk is exaggerated, pupils are normal and prompt, nystagmus is marked here, and this last symptom is never found in tabes. Lightning-pains are scarcely ever seen, since there are generally no sensory symptoms. Speech is staccato, and there is intention tremor. Confusion may arise when the lesion is in the lumbar cord, since the ataxia now will be combined with loss of knee-jerk; yet even here care will make differences apparent, for the upper extremities will have an exaggeration of the deep reflexes, and the characteristic nystagmus, intention tremor, or staccato speech, one, if not all, will be present.

General Paresis. This disease is at times so similar, that it has often been considered an upward extension of a tabes. The etiology is the same, except that if the history is searched, it will be found that the brain-worker, as a rule, becomes the paretic, while the mechanical worker has tabes as the reward of his syphilis. Knee-jerk may be lost in both, and the pupillary changes are the same, and both may be ataxic, but the paretic often has an exaggeration of the knee-jerk at first, while the tabetic never has anything but some grade of loss; pains are absent from paresis, usual in tabes, and if in paresis at all, they are spinal. The mental state of paresis is most distinctive, since it is an exaltation, or a deep melancholy with a terminal dementia, while the tabetic is only moderately apathetic, or reduced in mental acuteness. Paresis has a marked facial and bodily tremor, and slurring speech; both are wanting in tabes.

Paraplegias. At the first glance, the paraplegias are similar to tabes, but the differences are definite. In the ataxic form with a pronounced degeneration of the posterior columns, the difference is that while the patient is actually an ataxic, he has no pains, and the deep reflexes are exaggerated. In the spastic form, not only are the knee-jerks exaggerated, but there is ankle clonus also, and there is no ataxia, but in its place there is much weakness. In cases arising from a complete division of the cord, there is absolute paralysis in all parts of the body below the lesion, while such a loss of motion is not found in tabes, up to the last moment. There are changes in sensation in definite areas in tabes, while they are absent in the spastic, and often in the ataxic forms of paraplegia.

Cerebellar Tumor. Both this and tabes have ataxia, and occasionally the knee-jerk is absent in both, but as a rule the latter is exaggerated in cerebellar tumor. There is a tendency to fall in both, but it is much more of a drunken reeling in most cases of cerebellar tumor, and there may be jactitation of the feet. The ataxia of tabes pertains to the erect position, while it is just as evident when the tumor patient is lying down. He is much more liable to choked disc, his head is retracted, and he has occipital pains. The tabetic alone has the Argyll-Robertson pupil, lightning pains, sensory and bladder disturbances.

Syringomyelia. The lower limbs are generally paraplegic, but they may be ataxic, and the disease is generally seated in the upper part of the trunk, and in the upper limbs, which are usually spastic. Characteristically, with otherwise normal sensibility, the loss of the sense of temperature is the primary symptom. Muscular atrophy, painless whitlow, necrosis of the phalanges, and lateral scoliosis are present in syringomyelia, and are absent from the symptomatology of tabes.

TREATMENT. The whole subject of treatment has had to be revised in view of the discovery that the spirocheta pallida is the cause of this disease. Attention is now directed to the developement of a method of destroying this microörganism without injury to the tissues of its host. This is difficult for two reasons, the first of which is the fact that the spirochetæ at this stage have retreated into the deeper parts of the nervous system, and the second is that it has been demonstrated at the Rockefeller Institute, during the studies of meningitis and poliomyelitis, that medication introduced into the blood is barred out of the central nervous system by the functional activity of the choroid plexus. It was also discovered that medication must be renewed frequently, on account of the rapid elimination of foreign substances from the cerebro-spinal fluid. Swift and Ellis finally devised the following method for bringing salvarsan into immediate contact with the central nervous system with the minimum of danger to the patient. The patient receives intravenously a normal dose of salvarsan. One hour after the administration, a suitable quantity of blood is withdrawn, and allowed to clot. The next day the serum is poured off, centrifuged, and heated to 56° C. for half an hour. Lumbar puncture is now performed, and 25-30 c.c. of cerebro-spinal fluid is withdrawn, and the amount exactly replaced by the treated serum. This treatment is repeated every ten to twenty days, until the fluid shows a normal lymphocytosis, 2-4 to the c.m. This treatment has been extensively employed, and numerous modifications of it. Some patients have shown wonderful improvement, amounting, according to some reports, to practical cure. From such a happy condition there are all grades of result, even to apparent injury from the treatment. In drawing deductions it should be remembered that we are reasoning about an almost incurable disease, that the point of attack is a most sensitive structure, and that naturally some time must elapse before an unimpeachable technique, and a sure criterion for the selection of cases adapted to the treatment can be worked out. At the present time it can be said that the treatment appears to be theoretically correct, and that some cases have been very greatly benefitted, although no one can as yet be sure of the permanence of the improvement. If, after this treatment, the former invalid can perform hard manual labor, there is little need for after-treatment, but such cases must naturally be rare, and therefore other measures, which up to this time have been the whole of our rather feeble armamentarium, must still be employed.

Hygienic Measures. Improvement of the general health is a "sine qua non" for benefit from any form of treatment. Plenty of sleep is one of the most fundamental requirements. Ormerod insists that rest in bed for a period of from four to six weeks is in itself of great benefit. (This is the exact contrary of the results in multiple sclerosis, and the spastic forms of paraplegia, where it may result in making an ambulatory patient into a bed-ridden invalid.) Food should be carefully selected, and stimulants should be restricted, as well as tobacco, but the moderate use of either of these to one addicted to them probably has little direct effect upon the disease, but if they are objectionable from their collateral effects they should be stopped. There should be immunity from mental and physical strain. This latter should be estimated by the state of the pulse, and the reaction of the symptoms of the disease, since the patient's ability to estimate fatigue has been lost. See that the bowels functionate properly, and that the skin is active. Squier says that the first requisite is to keep the bladder sterile, if its function is disturbed. The patient is to be instructed concerning the management of the incontinence. After urination a catheter should be passed, residual urine, if present, must be drawn off, and a measured quantity of a mildly irritating fluid (Argentum nit. 1-1000) passed into the bladder, and the patient is told to make an effort to void it. Efforts are to be made at the same time each day, and the bladder emptied to the last drop, and stronger solutions, from one to two grains per ounce, may be used. The mild grade of tenesmus induced acts as a tonic to the contractile power of the bladder. Vesical spasm is best treated by the passage of cold sounds. Wachsmann has found, at the Montefiore Home, that lumbar puncture has benefitted bladder troubles, and in some cases has helped incontinence. It was also demonstrated that superficial massage was of little use, and that deep massage tore the muscles. A mild climate certainly conserves strength, and a change of air is often of great benefit, but let there be plenty of fresh air at all times. The patient should exercise, but with moderation, for the reason that fatigue may be incurred so easily.

Hydroptherapy has been used to a very great extent in the form of cold douches to the spine, steam baths, hot needle baths, and sponging the spine with alternate hot and cold water. So long as the reaction is complete and prompt, the stimulant baths are useful, but concerning the hot baths there is considerable difference of opinion. They are generally used for the relief of pain, and are fairly efficient for lightning pains (Oppenheim says they relieve at first, but aggravate finally), but they are of no use for crises, for which they are often employed.

Carbonic Acid Baths. Jean Heitz thinks that great benefit has come from carbonic acid baths to the number of 20 to 25, as they give them at Royat. It was shown in general condition, improvement in superficial and deep sensation, plantar sensation particularly; analgesia disappeared along the course of several nerve-trunks; stereognostic sense and that of position became more accurate, and ataxy less marked. He thinks that it acts by tonic effect on the myelin sheaths, and that it will keep a patient at his avocation longer than the Fraenkel movements. In the pre-ataxic stage the patient should practice the movements in which he shows deficiency, while if in an advanced stage, he should be given a full course of the movements prescribed by Fraenkel. Fraenkel (Berliner klinische wochenschrift, June 5th, 1905) says that his teaching in regard to exercises has been a failure, because overdone. Restrict the patient to two sessions a day of not more than 5 to 15 minutes each. The patient has lost his perception of fatigue, and so his feelings are no guide. The trunk muscles are the originators of the acts of locomotion, and exercise of them, and re-education of them are valuable, and have reduced the number of cases of atony of the abdominal muscles, and of the intestines, and also of cystitis. Weakness of the sphincters is best treated by the method of Brandt. While lying on the floor, or a hard bed, the patient should abduct and adduct the knees successively against pressure, and at the same time should vigorously draw up the pelvic floor. This procedure should be repeated about twenty times daily. Massage of the perineum is also of value.

Electrical Treatment. The best treatment, where galvanism is used, is to give an ascending current along the spine, placing the negative electrode on the back of the neck, or just under the ears, and the positive stabile in the lumbar region, continuing the current from ten to fifteen minutes with a strength of from 10 to 25 milliamperes, painting the spine with iodine before each application. Then place the anode on the abdomen, and the cathode stabile on one segment after another, taking about twenty minutes for the application. This will relieve very greatly the lightning-pains. Richardson recommends a cathode 1½ inches wide by 5 inches long, bridging the spinous processes, but not in contact with them. It is the active pole, and should be placed over the cervical spine. The anode is the other electrode, at least 3 to 5 inches long, and placed upon the sacral region. A current of 15 to 30 milliamperes is passed for three minutes, then turned off, and the active pole moved its length downward, and the same current used for the same length of time. The process should be repeated till every part of the spine has been covered once, and in bad cases twice, by the treatment. The strongest current should be used that can be borne, and that is usually about 30 milliamperes. Static electricity is very effective, if severe sparks are drawn from the spine and roots of the spinal nerves for at least three to five minutes; then milder sparks from various parts of the body following down the trunks of the nerves. Draw sparks from the bottoms of the feet for from two to three minutes, which reflexly affects the spine, improves the conductivity of the nerves, and has a great effect in temporarily relieving the ataxia. In the early stage galvanism helps, but static irritates; in the second stage where there is slight ataxia, galvanism is still the better. Where locomotion is very difficult, and the case is complicated by interference with the functions of various other organs, static is superior to galvanism. Many see a profound stimulation from the high-frequency currents in use to-day.

Counter-irritants have been used for a long time, but whether they act by suggestion, or really cause an increase of circulation to the part, I am unable to say. In some way or other they often seem to do good. The best method is the application of the cautery of Pacquelin. Bring it to a white heat, and make dots along the spine over the lateral processes. If it is at white heat, the application is quite devoid of pain. If nerves are affected, it may be applied along their course. In the use of the cautery it is best to repeat it at such intervals as not to make the skin an open sore, and give 6 to 10 applications, and then wait a month or two before making another series of applications. Nutrition of the spine is increased by flagellation of the back with a scourge, made by attaching a broad, stout piece of leather to a short handle. This may also be used to advantage on the soles of the feet, and the palms of the hands, and also on any anesthetic spots. Schwab and Allison say that the tabetic foot is a pronated foot. The effect of this pronation leads to muscular strain on the ankle, knee, hip, and spine. This, with the hypotonia, tends to break down the long arch, and makes a faulty apparatus with which to walk. If then we remedy this defect with a proper shoe, and make use of the Fraenkel method of re-education, we can give a great deal of aid to the tabetic.

THERAPEUTICS. The indications and clinical reports for the use of remedies are in many cases to be viewed with a great deal of suspicion, since a careful analysis will develope the fact that many of the so-called ataxias are due to other conditions than lack of sensory innervation, and many of the paralyses are distinctly spastic, while, on the other hand, pains give every indication of having been caused by rheumatism, instead of having been caused by irritation of the posterior nerve roots. Granting all this, and allowing that many indications are *sub judice,* there is no doubt that by the careful selection of remedies according to the rule of similars, many cases upon apparently slight indications have been greatly benefitted, and, while perhaps not cured, have been insured long periods of remission from painful or disabling symptoms. Physiological medicine, except in the one department of the relieving of pains, has not been any more favorable, and in my own experience seems to have been far less beneficial to the patient. Another point should be mentioned, and that is that remedies having a toxicological basis like Lathyrus, Secale, and Æsculus, have not given the amount of relief which might hopefully be expected.

Æsculus hip. This drug has produced in sheep an absolute tabes dorsalis verified post-mortem. It gives rise to a general paralytic feeling, with great weakness and lightning-like pains in the lower extremities. A characteristic symptom is a spasmodic contraction of the rectum, and if present in the disease should be an indication for the use of this drug.

Alumina. Apparently acts especially upon the muscle-spindles, and therefore we find anesthesia and paresthesia of the soles of the feet, and also quite generally distributed over the body, and even in the face. The use of the limbs seems to increase the subjective sensations of numbness; constipation is marked. The mental change is one of apathy.

Angustura vera. Is one of the doubtful remedies, since its symptoms are those of a spastic instead of an ataxic paraplegia, but O'Connor reports the relief of lightning-pains in two cases.

Argentum nit. The same criticism of this remedy has been made as of the preceding. Its ataxia seems far more dependent upon some form of multiple neuritis, or ataxic paraplegia, than upon tabes, but Allen considers that some cases have been cured by it.

Belladonna. Indicated for lightning-pains which have the characteristic of suddenness of onset and disappearance.

Cadmium met. The toxicological reports of this remedy show that it produces degeneration of nervous tissue, and its indication is cutting and lancinating pains like Zinc.

Carboneum sulphuratum = Carbon bisulphide. The use of this remedy is based upon its toxicology, reports of which are rather extensive, since it is a universal solvent for rubber. There are marked formication and numbness in the hands, with pains in the lower limbs, with cramps, trembling, and ataxia, the causation of which is doubtful. There are some clinical reports of its efficacy in the treatment of lightning-pains.

Causticum has been suggested, as its particular action is upon nerve-cells, which is the point of attack in tabes, but the paralysis which it causes has not the distinctive marks of tabes. There is a condition of tension in the muscles, instead of relaxation, as we should expect from the pathology, but there are paresthesias of the extremities, and it may prove to be useful in some cases.

Chromium sulph. has given laboratory grounds for its applicability.

Colchicum. Certain cases of tabes are marked by pains only, and with very few symptoms of motor disability. Cases are known to be precipitated, at least apparently caused, by exposure to cold and wet, and in such cases Colchicum should be useful.

Cuprum ars. and met. are marked by muscular cramps and lightning-pains, and have often been very efficient in relieving these symptoms in the early part of a tabetic seizure. There is a great tremulousness of the whole body on attempts at walking.

Duboisia. Has weakness in the limbs, and heaviness in them, with pain in the lumbar region, and a great deal of ataxia. If diplopia is found in such cases, this should be a good remedy.

Fluoric ac. For types with great sexual excitement, and with all symptoms excited by cold and wet. There is much paresthesia and subjective numbness in all the limbs.

Gelsemium has been considered to be a remedy for functional rather than organic conditions, and the tremulousness, neuralgic pain, and cramps in the muscles are irritative symptoms. If hyperesthesia of the surface is a marked symptom, which is unusual, Gelsemium will avert the anesthesia which will be the result.

Graphites has no particular grounds for use expect as an absorbent, but like some other remedies has been credited with cures.

Kali brom. It has great ataxia, and loss of perception of touch, pain, and temperature. It is considered to also produce an increase of sexual appetite, priapism, but without ejaculation.

Lachesis has been credited with cures, but the symptomatology shows a decidedly spastic form of paraplegia.

Nitric ac. is marked by burning superficial pains, *plaques douloureux*. Some cases of cures from its use have been reported.

Nux moschata has been held in great repute. Lippe marks it as his first remedy. The limbs are painful, with an aggravation in cold and damp weather, and there is a numbness in the limbs.

Phosphorus is Lippe's second remedy, and should be thought of in cases where, with great sexual excitement, there are burning and formication along the spine. There may be flashes of light before the eyes, followed by optic atrophy. A sexual excitement in the beginning, or at some period, is a *sine qua non* for its use.

Picric acid has anesthesia and subjective numbness of the extremities, but its main indication is a high degree of priapism. All these cases presenting this symptom would indicate an involvement of the cervical portion of the cord. This is a very unusual localization, but if present, one of these remedies should suggest itself.

Plumbum. This remedy has been highly recommended, and is said to be applicable to advanced cases where there is great sclerosis. There are numbness in the feet, and convulsive movements in the limbs, and while therefore it seems to affect the whole cord, it may be useful in cases where constipation is a marked symptom, and paresthesias of the extremities.

Secale. There are absence of knee-jerk, fulgurating pains and ataxia. There is no doubt that eating spurred rye has produced degeneration of the posterior columns of the cord, and while the results of its use have not been satisfactory, either when used in material doses, by Physiological therapeutists, or in potencies by our own practitioners, it should still be kept in mind as a possible resort.

Silicea. Cures have been claimed for this drug, but no distinct symptoms can be stated, and it must be prescribed upon general constitutional conditions.

Strontimum carb. has some indications which point to girdle sensations somewhat suggesting the tabetic cuirass. There is a paralytic weakness in the limbs, but it is to be noticed that there are symptoms of tension, which might be from root irritation, which would be the source of the preceding symptoms also. There is a subjective numbness in the limbs, and peculiarly also in the face. All the symptoms are very much aggravated by cold, and ameliorated by warmth and wrapping. O'Connor mentions that one case was very much relieved of lightning-pains by it in the 30th, the indication being that they were only relieved when the parts were in hot water.

Syphilinum. This is suggested from the fact that the disease is the result of the action of a late toxin of syphilis. Favorable reports have been given of the action of this remedy in the higher potencies. The author's experience has been that marked remissions have certainly taken place during its administration.

The necessity for some method of relief of the lightning-pains and crises is so imperative, that all sorts of measures have been advocated, only a few of which can be detailed here, since one cannot hope to cure a radiculitis by remedies; the most that can be hoped is that we may jugulate the pain at the time. Hypodermatic injections of Morphine will sometimes be the only refuge, and some have said that in this condition there is no danger of forming a habit, but this possibility must be kept in mind. The most certain results are to be obtained by the use of the coal tar-products. In order of efficiency they are Antifebrine, Antipyrine, and Phenacetine. Antipyrine may be given in doses of 10 grs. hourly, during the period of most acute pain, but as the pain subsides it should be given only three or four times a day. Bartlett says that 3 grs. given hypodermatically will relieve the spasm of pain in three minutes, but never give more than 5 grs. as an initial dose, as it is extremely irritating to the tissues. Sometimes benefit will be received by giving a thoroughly triturated powder of Acetanilid one week, Antipyrine the next week, and Phenacetine the following week. Ten grains of Phenacetine with 1-60th gr. of Strychnia sulph. will sometimes give relief, when any one of the analgesics alone will fail. A teaspoonful of baking soda internally will sometimes stop the pains. Cannabis Ind. 1-2 to 1-4 gr. every three or four hours so long as the pain persists. For the superficial pains put chloroform on lint, and cover it with oiled silk, or inject Cocaine very superficially, for if it is injected into the deep muscular masses it will do no good.

Bladder and Rectal Pains. Young says, give

Suppositories.	Pulv. Opii	Gr. 1.
	Ext. Belladonna	Gr. ¼.
	Iodoformi	Gr. 3.
	Ol. Theobromæ	Grs. 30.
	Misce et ft. Suppository No. 1.	

Pritchard believes in the efficacy of Cacodylate of Soda by mouth.

Given Campbell finds that Chloride of Aluminum gives absolute relief from the pains. This occurs before the dose reaches 1-2 gr. This is given three times a day, largely diluted, after meals. Has been given constantly for over a year, without disturbance to digestion or general health.

Loeper and Oppenheim advise Belladonna and Atropine in gastric crises for its action on the vagus.

Exner ties off the vagus nerve on both sides, and does a gastroenterostomy to avoid trouble from gastric ulcer, which he finds very common in tabetics.

Cade and Lariche have applied Franke's method, and have exposed the intercostal nerves at fault in crises. The nerve is exposed throughout its entire length, and is then lifted on a hook until the traction tears it loose from the spinal cord. They pulled out the 5th to the 10th thoracic nerves on both sides, and the patient has had no pain since, and has gained over twenty-four pounds. This operation is done in place of laminectomy. Mouriquand and Cotte think this method is good, but claim that the pain will sometimes recur.

SPASTIC PARAPLEGIA.

PRIMARY LATERAL SCLEROSIS.

Definition. This is a chronic, progressive degeneration of the spinal cord, which occurs in adults, with a weakness of the legs which is accompanied by a stiffness of those members. Its occurrence, as a separate entity, has been doubted, but Erb, who first isolated it, later reasserted his original proposition. This symptom-complex, however, is generally an early stage of some other, more widely manifested disease.

The Symptoms of a Typical Case. An adult will gradually develope a weakness in the lower limbs, with an exaggeration of the deep reflexes, which condition will finally render the person helpless, on account of weakness and rigidity. There will be ankle clonus, and Babinski's reflex when the condition has become marked, but from first to last there will be no sensory changes.

Differentiated from Syphilitic Meningo-myelitis, Pseudo-paraplegic Rigidity, Multiple Sclerosis, Myelitis, Amyotrophic Lateral Sclerosis, Hysteria.

Age. It occurs in adults from the thirtieth to the fortieth years.

Etiology. In the pure form under discussion it seems to result solely from a selective action of an infection, or from traumatism. Cases have been isolated in India which were caused by poisoning by Lathyrus sativus, or Chickpea. The form seen in infancy, or early childhood, is due to prenatal disease, or natal injury, and is described under the heading of Little's Disease. A similar form to Little's type, but not developing until the person is of mature years, is supposed to be due to the late consequences of the same sort of lesion, and suffered natally, or pre-natally, although of so slight a degree that its effects are actually a sort of precocious senile degeneration. There is a familial type which may come on about the fifth year, or be delayed until the twentieth or thirtieth, and may affect different members of a family for many generations.

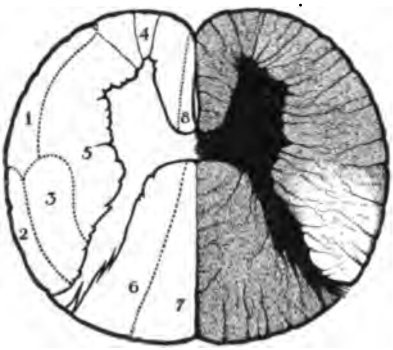


Figure 64. Spastic Paraplegia.

Observe that the degeneration shown in white in the right half is in the Crossed Pyramidal Tract (3), seen on the left half.

Pathology. There is a primary degeneration of the lateral colunms of the cord, with slight implication of the uncrossed pyramidal tracts. The same symptoms, so far as the lateral columns are concerned, may arise from compression of the tracts, but other tracts could hardly be expected to escape injury at the same time. The infantile, adolescent, and familial cases all arise from a primary degeneration of the brain, which is followed by a degeneration of these tracts, or from a deficient developement of them (agenesis).

Symptomatology. The first symptom to appear is a weakness of the lower limbs, but the patient is apt to explain that to his own satisfaction as being due to some acute condition. The rigidity of the limbs, which appears later, by its combination with the weakness, is apt to so embarrass his locomotion that he will seek an explanation from the physician. If then questioned, it will generally be found that he has been experiencing a sensation of weakness for a considerable time. Inspection at this time will reveal the fact that the toes of his shoes have been worn off in an unusual manner, his gait will be clumsy, and he will recall that he has been stumbling unaccountably of late. The deep reflexes will be found to be exaggerated, the knee-jerk is over-active, there is ankle-clonus, and a Babinski can be elicited. This consists in an over-extension of the great-toe certainly, and probably of all the toes, when the sole of the foot is stroked from the outside inward ending at the base of the great-toe. When the reflex is sluggish, the use of an instrument, with a somewhat sharpened extremity, may cause the reflex to appear more decidedly. The over-extension of the great-toe is all that is absolutely essential to determine the presence of the reflex. Gordon's reflex is the same over-extension of the great-toe, produced by firmly grasping the muscles of the calf. Oppenheimer's reflex is a dorsal flexion of the great-toe, produced by forcibly flexing the thigh upon the abdomen, and the leg upon the thigh. With the dorsal flexion of the great-toe there is apt to be a slight plantar flexion of the small toes. In the affected families there is usually some apparent cause for the degeneration, as alcoholism, syphilis, insanity, some one of the neuroses, or consanguinity. While the general symptoms are as detailed above, the upper extremities are rarely affected, but the lower extremities are most commonly involved, the feet presenting various forms of deformity, most frequently equino-varus. There is no wasting of the muscles, nor is the reaction of degeneration present. There is no loss of sensibility of the tissues, and pain is absent, except that there may be a little in the muscles, if the process is a rapid one. The limbs are the seat of painful spasm, particularly at night. The functions of the bladder and rectum are normally performed.

COURSE. It is a disease of slow progress, and it may be twenty years before the whole symptom-complex is evident. It may come to a standstill at any stage, or, after a long remission, again progress. For a long time, a spastic paralysis of the lower limbs may be the only departure from health, but in the full-developed disease, the arms finally become involved in a similar manner, and at times the face shares in the paralysis.

PROGNOSIS. Cures of definitely outlined cases are unknown, but the progress is so slow, remissions are so common, and the symptom of spastic paraplegia is so often only the preliminary stage of some other disease, that a definitely bad prognosis is uncalled for. If, after a time, we are convinced that it is lateral sclerosis, we can assure our patient of the slight danger to life, and a possibility of only a relative amount of disability. In severe and widespread cases, we can only hold out a hope of the continuance of the present condition.

DIAGNOSIS. This rests upon weakness of a moderate degree, spasticity (especially of the lower limbs), exagerated reflexes (deep), absence of sensory symptoms, or of disturbance of the sphincters.

DIFFERENTIAL DIAGNOSIS. Syphilitic Meningo-myelitis. This gives rise to a spastic paraplegia, but it comes on abruptly, and the degree of paralysis rapidly increases. It does not confine itself to the lateral columns, but gives rise to pains, and other changes in sensation, and disturbs the functions of the bladder and the rectum. It has a grave prognosis.

Pseudo-paraplegic Rigidity. This is a condition found in children who have suffered from rickets, laryngismus stridulus, and the so-called hydrocephaloid state. It begins in the hands instead of the feet, and is often confined to the hands and arms. It gives rise to spasms, instead of a relative rigidity, and attempts at reducing it by extension give rise to pain. It is also intermittent, and of transient duration.

Multiple Sclerosis. This is the disease for which spastic paraplegia is most often an initial stage. It may begin, and remain, for a time, a simple spastic paraplegia. Some sensory symptoms are possible, however, but more especially the early advent of some cerebral symptoms, vertigo, ocular palsy, or nystagmus, will differentiate the two conditions. Mental changes are common in multiple sclerosis, and are unknown in spastic paraplegia. If typical, multiple sclerosis is not at all confusing, since nystagmus, scanning-speech, intention tremor, and ataxia are not found in spastic paraplegia.

Compression Myelitis. This produces a spastic form of paraplegia, and therefore the initial periods of both may be similar, and this points out the necessity for conservatism in the diagnosis of spastic paraplegia. Alike at first, they very speedily diverge, and myelitis alone has root-pains, pains and hyperesthesia of the spinal column, and of areas of skin on the abdomen, and often on the legs, and girdle-sensations. Pain in the spine is often an initial symptom in compression myelitis, while it is never present in spastic paraplegia. The bladder and rectum are disturbed, there are often bedsores, and deformities from contractures are liable to occur.

Amyotrophic Lateral Sclerosis. This is a spastic form of paraplegia, but while the lower limbs are spastic and paraplegic, there is generally an early atrophy in the muscles of the arms. Atrophy occurs finally in all cases, and becomes very marked in the upper or lower extremities, or both.

Hysteria. This may exhibit a spastic form of paralysis, but sensory symptoms are always present, and generally of a distinctive sort. True clonus is not found in hysteria. The rigidity of hysteria is peculiar, in that the efforts to reduce it will be antagonized by a fixation of the extensor, as well as the flexors. The leg is steadied, as it were, in a determined position. When it succumbs to irresistible pressure, it gradually gives way, while the rigidity of spastic paraplegia is "lead-pipe," i. e., after a short period of resistance it suddenly doubles up, and is perfectly flexible. Chloroform narcosis will also render the hysterical rigidity perfectly flexible.

TREATMENT. The main reliance should be upon hygiene, nutrition, and stimulation of the nervous system. The patient should therefore lead a regular life, and, while using all means to promote muscular nutrition, should avoid over-exertion. The diet should be rich in fats, and cod-liver oil, and the petroleum emulsions, with or without the aid of hypophosphites, should be a part of his diet. There is a very great divergence of opinion as to the amount of rest that should be taken. Some advise a month's rest in bed as a preliminary to other treatment, and assert that it has started some cases toward remissions, who under other methods had been steadily becoming more and more incapable. On the other hand it is the observation, in places where large numbers of such cases are treated, that a month's entire rest may make a bed-ridden invalid of a person who was previously able to go about enough to earn his own living. The weight of opinion is in favor of moderate exertion, and the practice of the same exercises as are recommended in tabes. The aim of these exercises is the re-education of the spastic limbs, and has strong testimony in its favor. Stimulation by faradism and strychnine is condemned by all but a few. Its advocates acknowledge its theoretical danger, but urge occasional brilliant results. Hammond (Medical Record, Feb. 5th, 1910) says Strychnine is always bad. The relief of the spasticity is sometimes accomplished by warm baths, but still more efficiently by the Turkish-bath, or sweating, or warm or hot douches to the spine and limbs. Massage should be given in an upward direction, while the limbs are held to such a degree of extension that the muscles are tense. This is also good for the relief of the spasms of the muscles at night, which are so painful. For this is also used the spinal ice-bag. The warm bath should be from 90 to 95 degrees. One author, copied by many others, has said that galvanism is useless, and faradism is positively harmful, but King says cures reported of the early stages were too early to allow the diagnosis to have been established; it is, however, a palliative for the spasticity of the muscles. The strong static spark over the spine and nerve-roots is beneficial, but not over the muscles, except when paresis has become marked. It will then relieve by invigorating them, as will faradism. This is especially true of the quadriceps extensor. Galvanism may be used over the spine for eight minutes, with a descending current of twenty to twenty-five milliamperes, electrodes three inches in diameter. Put the anode on the abdomen, and galvanize each segment with the stabile cathode for three minutes, following with two or three interruptions on each segment. To relieve spasmodic rigidity in muscles, use the positive with a long, narrow electrode over the muscle, retaining the cathode on the spine. Raise the current gradually to ten milliamperes, and allow it to remain there for two or three minutes, and then as gradually reduce it. Go over every set of muscles in this way. Interruptions over motor points should not be made except very late in the disease. Steavenson of London places patients in a bath to which the cathode is attached, the anode being on the spine between the shoulders. Ten to fifteen milliamperes are given, for from fifteen to twenty minutes. Some, he says, are much invigorated, and the spasms reduced, while many are not helped.

Foerster of Breslau, in 1907, recommended section of the sensory roots to the spastic muscles, on the theory that the spasticity was due to the over-activity of the motor-cells, due to lack of cerebral control. In the Deutsche medizinische Wochenschrift for May 12th, 1910, Hevesi reports success in the case of a spastic diplegic girl of eleven years. She was laid on the face, the lower end of the spinal canal was opened, the roots of the cauda equina were lifted on a blunt hook, the nerves corresponding to the 2nd, 3rd, and 5th lumbar, and 2nd sacral on the right side, and the 4th lumbar, and 1st sacral on the right were resected from 2 to 6 cm. Frazier (Journal of Surgery, Gynecology, and Obstetrics, Sept., 1910) describes the technique at length, and reports on the literature to date. Taylor does the same in the N. Y. Medical Journal, Jan. 29th, 1910.

THERAPEUTICS. The outlook from remedies is well-nigh hopeless, but, since some cases recover whose symptomatology is distinctly that of spastic paraplegia, it is well to do all that is in our power to possibly abort a disease which is incurable in the chronic stage. Since this is not a cellular disease (we know that cells once destroyed are never regenerated), it may be possible that patches of sclerosis may be absorbed. Strychnia phos. 3x is sometimes of benefit, and if such a potency aggravates the symptoms, a higher one may be useful. Mercury is always a possible remedy in sclerosis of nervous tissue. Many other remedies have been advised, but in many of these sensory symptoms are too prominent for them to be regarded with favor. The distress from muscular twitchings, especially of the legs, and most prominent at night, is possibly to be helped by Cuprum met. 6x. Argentum nit. has indications of a simple spastic paraplegia, with the weakness, heaviness, and stiffness in the legs, but there are also cramps and rigidities in the muscles at night. There is also a tearing pain in the muscles and nerves which has no place in the symptomatology of spastic paraplegia. Paraplegia with a simple weakness may find its simillimum in Causticum. It is a right-sided remedy. While Belladonna (Atropia sulp. as well, of course) has a great many sensory symptoms in its pathogenesis, it nevertheless should be thought of for the relief of the muscular twitchings. Conium is probably a functional rather than an organic remedy, as is Hyoscyamus, but may be of use. Lathyrus has an antipathic basis for its use, as it produces, when eaten, a spastic form of paralysis, with exaggeration of the reflexes, without sensory symptoms, except muscular cramps. It has vasomotor influence, which is shown by a blueness and coldness of the extremities. A cure from its use in the 3x is reported; the case, however, was probably a myelitis, since the patient presented the symptom of a "girdle sensation" as though a cloth wrung out of cold water were wrapped about his waist. I have seen good results from its use, however, and should always think of it as a possible remedy. Manganese presents many sensory symptoms in its pathogenesis, yet Searle quotes a case where hourly doses of the Binoxide 3x gave speedy relief to acute symptoms simulating spastic paralysis. Phosphorus has paralysis, but it is always marked by sensory symptoms. Plumbum has the same. Ustilago maidis also has pains. Zinc has heaviness, stiffness in the legs, nervous movements when lying down, and in sleep, but there is marked sticking and burning pain. The stiffness and spasm seem to result from the neuritic condition, more than from the central condition. There is paralysis from exhaustion, and auto-intoxication. There is constant reference to pains in the symptomatology. Nux vomica is advised, but the symptomatology has many references to disturbed sensation, pain, numbness, and formication. Graphites, Hepar, and Silicea should be thought of for their efficiency in promoting absorption.

Of the physiological remedies, a trial of Bromides, and other sedatives, has developed a rather general agreement, that Antipyrin is the best.

ATAXIC PARAPLEGIA.

Postero-lateral Sclerosis.

Definition. It is rather common among organic spinal diseases to find a paraplegia which involves both the lateral and the posterior columns. This Gowers considered to be a definite symptom-complex, exhibiting a spastic paraplegia combined with an ataxic unsteadiness, which latter might at first be the dominant symptom. To this he gave the name of Ataxic Paraplegia. It has never met with unqualified acceptance. Oppenheim decided that this apparent complex was generally a stage in the evolution of tabes, but that occasionally the pure type was to be found. Dana says that most of these cases would be correctly classed as locomotor ataxia, multiple sclerosis, or as some form of chronic myelitis. There is a combined degeneration resulting from anemia, which is quite similar in localization.

The Symptoms of a Typical Case. There is a spastic paraplegia of the lower limbs, with an increase of the deep reflexes, but also with some sensory changes. Later a pronounced ataxia appears, which is followed by a diminution, and perhaps a total loss of the deep reflexes, some symptoms of bulbar invasion, optic atrophy, nystagmus, and mental deterioration.

Differential Diagnosis. It is to be diagnosed from Tabes, General Paresis, Multiple Sclerosis, Spastic Paraplegia, Chronic Myelitis, Family Ataxias, and Cerebellar Tumor.

Heredity. The pure type is said to be hereditary in one-tenth of the cases.

Age. Usually from thirty to forty, but it has occurred at fifteen, and as late as sixty-one.

Sex. Males predominate.

Etiology. It is supposed to arise from a very wide range of causes. It was formerly supposed to develope from both tabes and paresis, from the fact that, at times, in their evolution this symptom-complex would appear. It has followed an attack of myelitis, but in the more modern view, it may be considered in these cases, that it is only a stage in a chronic myelitis. Lepto-meningitis has also been alleged as a cause, when in its extension it has set up a marginal form of myelitis. Specific and general intoxications have caused it, like pellagra, and syphilis, although it follows syphilis as infrequently as tabes does frequently. It has followed directly upon muscular over-exertion, and particularly when exposure followed such over-exertion. Simple refrigeration, physical shock to the spine, and sexual excesses are all given as causes by Gowers. It has arisen without any assignable cause, and therefore there is strong probability that embryonic deficiency, or localized vulnerability lies at the root of all cases.

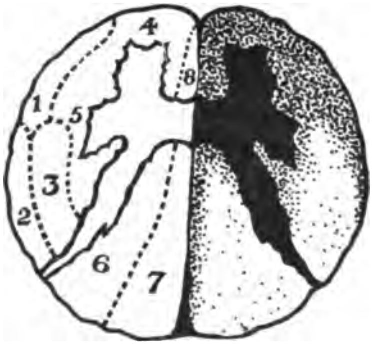


Figure 65. Ataxic Paraplegia.

The degenerated portions appear as white areas in the right half. They may be identified by reference to the schematic left half. They are the column of Goll (7), of Burdach (6), the Crossed Pyramidal tract (3), and the Direct Cerebellar (2).

Pathology. It is a sclerosis resulting from endarteritis of the spinal vessels, and varies in vertical extension according to the duration of the disease. The pia has been found adherent to the posterior and lateral aspects of the cord. The sclerosis is usually confined to the distribution of the posterior spinal arteries. The dorsal region is the first attacked, and the lumbar region may be spared to a great extent. In long-standing cases the region of the anterior arteries is invaded to some extent, and the column of Clarke, and the anterior spinal roots have been degenerated, but complete destruction of any definite tract is rare, and the posterior root-zone is very rarely involved (opposite of tabes). The complete picture is a degeneration of the column of

Goll throughout its whole extent, column of Burdach above the lumbar enlargement, crossed pyramidal tracts only slightly, direct cerebellar very severely, while the column of Gowers often escapes entirely.

SYMPTOMATOLOGY. As has been said, it is a combination of an ataxic and a spastic paraplegia, and not at all uniform in the proportion of either form of disability, but usually it first becomes pronounced as a spastic form of paraplegia, complicated by a very definite ataxia. Since the direct cerebellar tracts are so uniformly and so severely injured, we are sure to find a high degree of ataxia and incoördination, and the well-nigh universal sclerosis of the posterior columns indicates that sensory disturbances will be prominent. We are sure to encounter some degree of anesthesia and paresthesia, but lightning-pains are almost unheard of. Some sacral pain is diagnostically present, and is also apt to be in the legs. The knee-jerks are exaggerated at first, but generally diminish at a later stage. The knee-jerks are preserved, because the removal of inhibition by disease of the lateral columns allows stimulation of anterior horn cells, and production of the reflex, even with impairment in some part of the arc. Ankle-clonus is generally present, but Romberg's sign and an ataxic gait are always present to some degree. The disease usually ascends the cord, and the upper extremities become spastic, and also incoördinate, and there are irritation symptoms of the bulbar nuclei, i. e., twitchings of the face and tongue, and thickening of speech, and some ocular symptoms, viz., reflex accommodation being lost, that to light remaining, optic atrophy (syphilitic cases), and nystagmus on movement. The mind is rarely affected, and if so, only to a slight degree of memory defect. Owing to the involvement of the lateral columns, there is a degree of muscular weakness proportionate to the involvement of those tracts, and a tendency in all muscles to cramp-like spasm on use of them. There is no marked atrophic change, and while cystitis may occur, it is from retention owing to the spasticity of the sphincter. Sexual power is often lost early in the disease. The combination of symptoms does not follow such a regular order as is common in system diseases, and while those just enumerated may be present, many may be lacking, some may be added (arising from the primary and exciting disease), but, in order to make the disease come under these captions, there must be some combination of spastic paralysis and ataxia. In a case now under treatment there is a history which may be illuminating. A man probably acquired syphilis at the age of about thirty-two, and since then has had repeated prostrations, which have been attributed to nervous exhaustion from business cares. In 1890 he had a suddenly developed ptosis of the right eye, lasting thirteen weeks; at the same time retention of urine, some vertigo, weakness of the lower limbs, and marked ataxia. At the present time he has both knee-jerks exaggerated, the left more than the right, ankle-clonus on both sides, Babinski on the left side, cremasteric reflex is present on both sides, but more pronounced on the left. The radial jerks upon both sides are exaggerated. The left pupil is larger than the right, and there is doubtful Argyll-Robertson pupil. Romberg is very evident. A numbness began in the glutei, and there was a doubtful sensibility of the ball of the left foot. There is no tactile anesthesia of any region, but subjective over the whole of the legs, with girdle and cuirass sensations, and there is analgesia in the region of distribution of dorsal 4th to 12th inclusive.

COURSE. It is a disease of long duration, and not directly inducing a fatality, but the disability does not allow the patient to lead an active life, and therefore he has a greater vulnerability than the normal person. There is a peculiar danger pertaining to any disease which prevents the proper emptying of the bladder, and so these patients are liable to succumb to cystitis, or pyelitis. Some of them, however, live out their allotted span of life. It is generally from one to two years before the disease becomes fully established, although there is great variation from the differences in the causative condition.

DIAGNOSIS. This rests upon the presence of marked ataxia with a spastic form of paraplegia, and the combination of these with some sensory symptoms, and an absence of any definite and immediate atrophy.

DIFFERENTIAL DIAGNOSIS. Tabes resembles it in the symptom of ataxia, and some of the eye-symptoms might be confused, the girdle-pains, disturbances of sensibility and of the sphincters, but the ataxia of tabes occurs with a lowered or lost knee-jerk, never with an exaggerated one. The eye-symptoms of tabes are an irregular or distorted pupil and some third-nerve palsies, but above all the Argyll-Robertson pupil, which is either absent, or very poorly marked in ataxic paraplegia. There is always some objective loss of sensibility in tabes, and it may be only subjective in ataxic paraplegia. Tabes frequently has lightning-pains and crises, while ataxic paraplegia rarely has lightning-pains, and never crises. The upper limbs are almost always spastic in ataxic paraplegia, and never in tabes. Tabes is not weak in definite muscles, while the ataxic paraplegic is conspicuously weak.

Multiple Sclerosis. This is principally distinguished by the presence of symptoms wanting in the other disease. The mind is weakened, and the patient is emotional. Instead of a halting and imperfect speech, the typical multiple sclerosis patient has scanning speech, nystagmus, and intention tremor. Sensory symptoms are almost always wanting.

Spastic Paraplegia. This has weakness, spasticity, exaggerated deep reflexes, and an absence of any sensory symptoms.

Myelitis. There are two types, acute and chronic. The acute is readily distinguishable by its rapidity of onset, while the chronic form may be very similar on examination. It, however, is more limited in its distribution, and therefore the range of its symptoms will be more restricted. It tends also to run a course limited in time, while ataxic paraplegia may remit, but never recedes.

Family Ataxias. These are very similar in symptoms in the form of Marie, so far as the exaggeration of reflexes is concerned. They are, however, peculiarly familial, are marked by a preceding mental defect, and are always earlier in life, except in the very exceptionally early cases of ataxic paraplegia. Here, however, we find in the family ataxias some nystagmus and speech defect as an early symptom.

Cerebellar Ataxia from Tumor. Here we find ataxia, but with it vertigo, vomiting, choked disc, and forced movements. While a late involvement of the pyramidal tracts may produce weakness and exaggeration of the deep reflexes, the weakness is never so marked as in ataxic paraplegia.

TREATMENT. When we make use of adjuvants, we must follow the same plan as we would follow in the treatment of a spastic paraplegia, and treat the sensory disturbances like a case of tabes. In view of the possibility that an intoxication is the exciting cause, elimination should be as perfect as possible, and the patient should be given the benefit of fresh air and sunlight. Massage is valuable in these cases, and if they recover, the re-education method of Fraenkel should be employed to reduce the ataxia.

Electrical Treatment. In the early stages the spastic symptoms are pronounced in the lower limbs, and they may be persistent. So long as this condition persists, strong static sparks over the spine, and at the points where the nerves emerge, will be found to help the spasticity. This treatment may be modified by giving galvanism on the spine as is recommended for spastic paraplegia. When the muscles become paralyzed, faradism may be used with more benefit, especially on the quadriceps extensor. When the sensory symptoms become evident, the static treatment of the spine should consist in drawing sparks from it for from three to five minutes; then the milder sparks from the affected parts of the body, especially over the trunks of the nerves, and then from the soles of the feet for two or three minutes. In mild involvement of the cord use galvanism with the cathode over the involved segments, and the anode over the abdomen, and an interrupted current. Another method is by including the segments between two electrodes placed upon the spine, and passing the sinusoidal current between them. When the static is used, the wave current is sometimes of benefit.

THERAPEUTICS. If the cause is syphilis, modern anti-syphilitic treatment should at once be instituted, while if it is a complication of a tabes, our attention should be focussed on that disease. The best general indication is to give those drugs which have been recommended for tabes dorsalis. In the selection, however, the combined character of the sclerosis should be kept in mind, and only those drugs should be prescribed in whose pathogenesis some spastic symptom can be found.

Ustilago. The symptoms seem to be more ataxic than paralytic, and there are pains and numbness. O'Connor believed that it might be of service, especially in women.

Benzinin dinitricum has symptoms of vertigo, ocular anomalies, numbness with paralytic sensations, and spasticity.

COMBINED DEGENERATION OF THE CORD IN COMBINATION WITH ANEMIA AND THE CACHEXIAS. PUTNAM'S TYPE.

DEFINITION. This is a rather rare type of sclerosis, first described by Putnam, and later by Dana. It is a combined sclerosis, but differs from the ordinary forms in the wider range of the symptoms at times, and in other cases by a peculiarity in their distribution. It is a product of profound anemic processes, but it sometimes precedes rather than follows the anemia.

THE SYMPTOMS OF A TYPICAL CASE. These are hardly capable of concise description, but it is safe to say that a person in adult, or late adult life developes paresthesia of the extremities, which is soon followed by symptoms of a combined spastic and ataxic paralysis, with a rapidly developing anemia, of a profound type. In about six months symptoms of constitutional degeneration appear, the brain may become weakened, and the person dies in about two years with the symptoms of pernicious anemia.

DIFFERENTIAL DIAGNOSIS. It is to be differentiated from Ataxic Paraplegia, Multiple Neuritis, and Locomotor Ataxia.

AGE. It is a disease of late life, most often between the ages of 55-65, next between 40 and 50, and Rothmann's case was 35 years of age, the earliest reported.

SEX. It has been stated that it is more common in males, but Dana's cases were female in the proportion of nine out of twelve.

HEREDITY. There are some grounds for believing that it is familial and hereditary.

ETIOLOGY. Pernicious anemia is generally the cause, but the effect of profound secondary anemias is often evident. It follows exhausting diseases, infections, and cachexias. Dana instances diarrhea, dysentery, shock possibly, lead poisoning, malaria, lathyrism, pellagra, and one patient had a malignant tumor, but syphilis is not a cause. Two out of six of Leopold's cases were tuberculous. While most of the patients exhibit the anemia, others develope the neural symptoms initially, and he therefore questions whether it may not, while truly an intoxication, be primarily caused by the presenile failure of some of the glands of the body. By their failure to functionate properly, the endogenous and exogenous poisons are no longer neutralized, or the same result may depend upon intoxications from the intestinal mucous membrane, which in some cases has been found to be atrophied. Linel, after a review of the literature, and the study of a case of his own, is unable to decide whether the disease has a vascular or toxic origin, and can only profess himself unable to find any reason for its selective attack.

PATHOLOGY. It attacks by preference the postero and postero-lateral white tracts of the cord, although it may attack any or all of the tracts (Gordon reported a case in which the Column of Gowers was affected). It may extend into the anterior horn cells; it may exhibit sclerotic areas in the brain, but does not affect the meninges, or the peripheral nerves; the anterior and posterior roots are slightly, if at all involved. The spinal changes consist of symmetrical areas of sclerosis, involving most frequently the posterior and lateral tracts in the upper dorsal and lower cervical region, but showing no predilection for any one system of fibres. It tends to spread into new foci so that the whole length of the cord may be implicated, and in some cases the cord shows softening and cavities. From these facts, Leopold (Medical Record, March 5th, 1910) considers the disease to be focal and not systemic. The blood-vessels are congested, and their walls show hyaline degeneration at times. The perivascular spaces contain leucocytes and granular material. Beside the neuroglia proliferation, rarefaction of the fibres, and many spaces are noted. Where they have disappeared sometimes small hemorrhages are seen, and the cells in the gray matter show pigmentation (Leopold). Some cases of years duration have on autopsy shown very slight organic change.

SYMPTOMATOLOGY. The symptoms are such as would be expected in a case of pernicious, or profound secondary anemia. Next we find such symptoms as would arise in a case of ataxic paraplegia, plus a variable group whose existence demands a degenerative condition of some other parts of the cord, and, in the later stages, of the brain also. Initially there are paresthesias of the lower limbs, or of the upper, or of all the extremities, which symptom is marked and persistent. Next we find a weakness of the lower limbs, with spasticity, and, as would be expected, exaggeration of the deep reflexes. There is ankle-clonus, and also Babinski's toe-sign.

At the same time there is marked ataxia, and consequent upon the rigidity of the limbs, and the presence of ataxia, which is cerebellar in type, the ability to walk is very seriously compromised.

There is generally a gradual onset covering many months, but one of Leopold's cases developed weakness and paresthesias in twenty-four hours. The paresthesia has rarely assumed a girdle-like distribution. Spiller reported a case where the pain was at the level of the third dorsal vertebra, and thence radiated. He also quotes the following typical case. The patient experienced at first numb, creepy feelings in the right leg, and soon after the left leg became similarly affected. One year later his gait became unsteady, and his feet felt numb. Four months later he could not use his lower limbs, and in a few months more he developed dribbling after urination. On physical examination it was found he could very slightly rotate the lower limbs inward at the thigh; otherwise the limbs were paralyzed. He could not even move his toes. The patellar reflex was entirely gone on the right side, even on reënforcement, and was very faint on the left. The Achilles reflex was nearly normal on both sides, and Babinski was present on both sides. The plantar reflex was greatly exaggerated on both sides, especially the left. Irritation of the left sole caused the leg to be flexed two or three times, and contractions occurred in most of the muscles of the lower limb. The cremasteric reflex was preserved on both sides. Perception of pain, and touch, and temperature was entirely lost in the lower limbs. Sensations of the same sort were diminished over the front of the trunk, below the nipple-line. Sensation was lost in irregular areas over the thighs. The movements of the upper limbs were free, and the hand-grasp was normal in both. Three weeks later there was a complete paralysis of the lower limbs, sensation absent up to the umbilicus, and just above this a band of hyperesthesia three inches in width. The grasp was weaker, and the patient complained of a numbness in the hands. These cases show strabismus, facial palsy, inequality of the pupils, and a slow response to light. The larynx is not always spared. Bulbar symptoms are reported, and several cases of double optic neuritis. While the spinal symptoms predominate, the mind is often affected. Shallow confusion is noted, with impairment of the ideas of time and place, with fabrication, and the narration of imaginary experiences, reminding one of Korsakoff's syndrome. There may be apathy, and melancholy, somnolence, or delirium, while some may be emotional, and hysterical.

Course and Prognosis. When the cause is syphilis the prognosis is good, otherwise the disease runs a very rapid course, being fatal in about two years, but remissions have been seen, so that cases have lasted as much as five years, and in view of these remissions Dana believes that cure may sometimes result if cases are seen sufficiently early. Hare says, "Curable, if the anemia can be cured." In some cases the causative anemia is so severe that the symptoms never reach a very definite developement.

Diagnosis. The diagnosis rests upon the discovery, by blood examination, of a pernicious or profound secondary anemia, combined with the symptoms of ataxic paraplegia. In cases where the anemia follows instead of precedes the paraplegic symptoms, it will be suggested by the addition of the ataxic syndrome of cerebral, ocular, and sensory symptoms.

Differential Diagnosis. It is to be diagnosed from ataxic paraplegia, which it most resembles by the wider distribution of symptoms, its more rapid progress, and as a rule by its evident etiology. Sensory symptoms are earlier and more prominent.

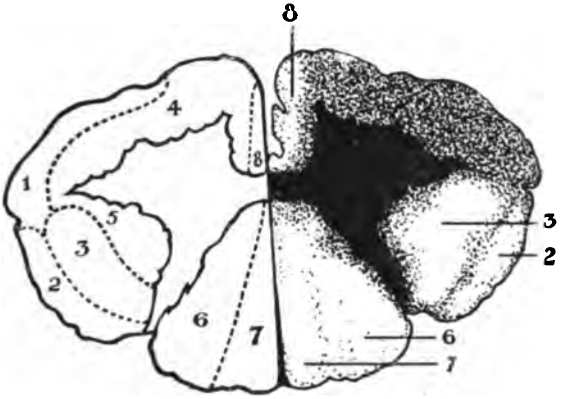


Figure 66. Spinal Degeneration in Connection with Anemia.

Degenerations (comparing white areas on right with scheme on left side) will be seen to effect column of Goll (7), Burdach (6), Crossed Pyramidal (3), Direct Cerebellar (2), and Direct Pyramidal (8).

Multiple neuritis is much more acute, muscular wasting is a prominent feature, there is much local tenderness and pain in place of a general paresthesia of the extremities, and the bladder and rectum are not involved.

Tabes. It differs from tabes in that the ataxia is cerebellar in type; it is a paraplegia, and not exclusively an ataxia.

Treatment. The initial treatment should be directed to the correction of the anemia, and since the spinal degeneration is a random one, we should also consider remedies which would be appropriate in a case of myelitis.

Electrical Treatment. First pass a current through the section of the cord where the hemorrhage has taken place. This may be begun much sooner than in cerebral hemorrhage; a week or ten days is sufficient time to elapse. The treatment of the peripheral nerves should be the same as in spinal meningitis, or myelitis. If anesthesia exists, the faradic brush may be used. The static spark has been very highly recommended, and the rules governing it should be the same as those given for spinal meningitis, or myelitis.

Physiological Treatment. This has been intestinal antisepsis, colon washing, combined with tonics, of which Arsenic is the most generally commended.

THERAPEUTICS. Arsenic is the best remedy under many conditions, and may be useful in all potencies, and also in the crude drug; Calcarea carb.; China; Secale peculiarly for spinal anemias; perhaps in material doses. Phosphorus; Plumbum.

FAMILY ATAXIAS.

FRIEDREICH'S ATAXIA—MARIE'S HEREDO-CEREBELLAR ATAXIA—HEREDITARY SPASTIC PARAPLEGIA.

DEFINITION. There are several forms of degeneration of the central nervous system which have the common characteristics that they affect young people, appear in successive generations of a family, and produce some type of ataxia as their prominent symptom. While not always clearly differentiated, there is a sufficient variation in the clinical pictures to permit their division into fairly well separated groups, viz., Friedreich's Ataxia, Marie's Heredo-cerebellar Ataxia, and Strümpel added Hereditary Spastic Paraplegia.

THE SYMPTOMS OF A TYPICAL CASE. A young person, somewhere between puberty and the early twenties, developes a clumsiness of gait, due to ataxia, and later all of his members become affected. This is progressive, and he finally becomes incapable of following any vocation on account of loss of muscular dexterity, and he appears to have an equal mental deficiency. The muscles finally atrophy, and contractures occur in the limbs, and also in the muscles of the spine. Sensibility, the sphincters, and the electrical reactions are very slightly disturbed, if at all, but while the deep reflexes are lost in the Friedreich type, they are increased in the type of Marie. Muscular contractures are trivial in this latter type of Marie, but chorea, optic atrophy, and double vision are peculiar to it, and the Argyll-Robertson pupil may be present.

DIFFERENTIAL DIAGNOSIS. The condition is to be diagnosed from Tabes Dorsalis, Chorea, and Multiple Sclerosis.

FREQUENCY. The disease is rare in any of its forms.

RACE. It is most common in America, and least common in France, if observed cases may be used as a basis (Dana).

ETIOLOGY. Investigation has shown that heredity is the most potent factor in its production, and cases have been traced as far as four generations. It is most often indirect; i. e., the parent has been neurotic, or alcoholic, rather than an ataxic; it has been known to follow tabes dorsalis, but in some groups it has been direct. It appears at times in consanguineous marriages. In a family where many children are affected it tends to attack the succeeding children at an earlier age, and Dana says with increasing severity. It affects more males than females, but is most apt to be transmitted in the female line. Children nursed by their mothers are more apt to develope it. Its appearance at about the age of puberty, or in the later cases at the entrance of a person into active life, shows that its victims are endowed with a nervous system too weak to endure the strain of active life, or even of physiological developement. In some cases the additional element of intoxication by some of the infectious diseases has seemed necessary for its appearance, and therefore it has been observed to follow typhoid, measles, scarlatina, rheumatism, syphilis, and possibly diphtheria.

PATHOLOGY. The common findings are areas of sclerosis in the posterior and lateral tracts of the cord, leaving clear spaces here and there. The direct cerebellar and Gowers' tracts are universally involved, and the posterior cell areas exhibit a sensory atrophy, this being most marked in the Column of Clarke. The medulla is usually immune, and the hypoglossal, and the optic are usually the only cranial nerves involved. The cerebellum is extensively sclerosed in the cases of the Marie type, and may be reduced to one-half or one-third its normal size, but the brain is practically unaffected. The posterior nerve-roots are greatly sclerosed, the anterior less so, and there is evidence of chronic leptomeningitis on the posterior aspect of the cord. The peripheral nerves are not as extensively affected as in tabes. The cord is shrunken and harder than normal. The histological change seems to be a gliosis.

FRIEDREICH'S ATAXIA.

AGE. Usually about puberty, but may arise in early childhood, or as late as twenty-five years of age.

SEX. Male cases are most common, but the inheritance is from the female side.

PATHOLOGY. The cord is diminished in size, it is degenerate, or undeveloped in all its tracts, and not only fibres, but cells also, are degenerated. The cerebellum is not always spared.

SYMPTOMATOLOGY. At puberty the patient is found to have become clumsy in his gait, and provokes comment among his companions on account of it, or he is constantly rebuked, at home, or in school, because he is continually dropping articles, or fails to write or to draw as successfully as his fellows. On account of this criticism he becomes bashful, and fails in mental grasp, apparently, and this may be the initial ground of complaint. If, however, he is carefully examined, no trace of mental dulling can be found at this stage. The musculature will now exhibit, on careful examination, an ataxia, most marked in the lower extremities, and there will be an apparent Romberg. If the test is made with both open and closed eyes, it will be found that the ataxia is as great under the one as under the other condition, showing that the fault is in the cerebellum. The result of this on the gait is that it is staggering and drunken, while in the spinal ataxia the gait is good in the early stages while the eyes are open, and the feet can be guided by vision. When the patient stands or walks there may be an irregular nodding or wabbling of the head, or grimacing of the face, which has been termed "nystagmus of tht face." These movements are the most marked in the head, neck or face. Strength will be found to be good, and therefore we can eliminate any paralysis. All tendon reactions are diminished and become abolished, and the knee-jerks soon become reduced to extinction, in a large majority of cases. The knee-jerks may persist until a late stage, but Babinski's sign is generally present. The sphincters are unaffected in both this and Marie's type. The muscles become the seat of contractures, and the arch of the foot draws up, producing "pes cavus," while the contraction of the muscles of the foot produce "hammer-toe." All the toes may be affected. This combination of contractures is quite a point in diagnosis. Late in the disease, the same tendency to contracture irregularly manifested, produces scoliosis, most marked in the dorsal and lumbar spine. The involvement of the cranial nerves, in the later stages, is often shown by the presence of nystagmus, and the involvement

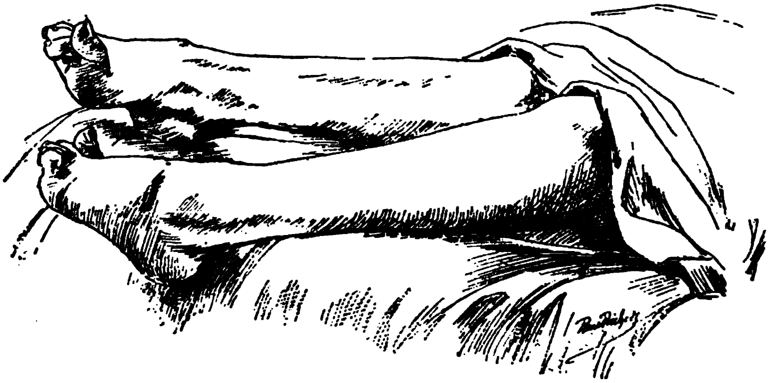


FIGURE 67. FRIEDREICH'S ATAXIA.

of the glosso-pharyngeal produces a slow, thick, clumsy speech "hot potato speech." The pitch of the voice may also vary from word to word, and advanced cases will show an actual mental feebleness. The general muscular innervation is so poor that the patient becomes generally shaky; this may be evident in the face when talking. In the early stages there is apt to be a chorea, and it may remain permanently in certain localities. Electrical reactions are usually only depressed, but in a few instances some reaction of degeneration has been found. There may be some fibrillary twitching. Sensory disturbances are rare, but some cases suffer at first from pains, and later there may be a decrease in sensibility.

MARIE'S HEREDO-CEREBELLAR TYPE.

DEFINITION. This disease is essentially a cerebellar ataxia.

AGE. Twenty to thirty, instead of about puberty, as is the case with Friedreich's type.

SEX. There is no marked difference in the victims, but it is usually transmitted in the female line.

PATHOLOGY. This has many symptoms in common with Friedreich's type of ataxia, but autopsies show a very different localization of the degenerations, or mal-developements. The principal loss here is a mal-developement of the cells of the cerebellum.

SYMPTOMATOLOGY. Many symptoms are common to both varieties, but in Marie's type the tendon reflexes are exaggerated, and not diminished, but the superficial are not affected in either, nor are the sphincters. In this type the ataxia is of a distinctly cerebellar type. The patient rolls and sways while walking, yet he can close his eyes, and stand with his feet close together, i. e., he has not the Romberg symptom. Chorea is more common and pronounced in this type. There may be a symptom similar to nystagmus, optic neuritis ending sometimes in optic atrophy and double vision, and the Argyll-Robertson pupil may be present. While common in Friedreich's form, clubfoot and scoliosis are rare in Marie's type. All family ataxias occurring late in life tend to the cerebellar type, and have increased reflexes.

COURSE. The course of all is very slow, some having lived for from twenty to forty years, suffering only in locomotion, dexterity, and some mental impairment, or non-development.

PROGNOSIS. Most authors assert that no cases have been cured, but life is not threatened by the presence of the disease. Friedreich's type may go on to complete helplessness.

DIAGNOSIS OF FAMILY ATAXIAS IN GENERAL. This rests primarily upon the developement of an ataxia in a young person, who can be proved to be free from a neuritis, the absence of the reaction of degeneration, and of sensory symptoms, the presence of mental deficiency, with marked ocular and speech-defects, and "pes cavus," hammer-toe, and scoliosis, will be additional factors in its favor.

DIFFERENTIAL DIAGNOSIS. Tabes is like in many respects, but the patient with hereditary ataxia is too young, his family is liable to show other cases, or is markedly neuropathic. He has no lightning-pains, no anesthesias, no bladder troubles, no visceral crises, no trophic disturbances, no loss of muscle sense, and except in a case of the Marie type, he has no Argyll-Robertson pupil. Here the presence of nystagmus, scoliosis, per cavus, and manus cavus are also positive indications against a diagnosis of tabes.

Chorea. Since chorea may be an .incident, or a complication, of both forms of family ataxia, the diagnosis must be upon broad grounds. In the history of a case of family ataxia, we find that the patient first became clumsy, shaky, and ataxic, all depending especially, and often solely, upon some defect exhibited in the lower limbs. The choreic exhibits the characteristic movements abruptly and initially, and always most evidently in the upper limbs. Granting the condition to be atypical, or impossible of verification, there are symptoms present in the family ataxic which are absent from the symptomatology of the choreic. He has no nystagmus, nor scoliosis, no clubfoot, no hammer-toe, and the reflexes are not persistently diminished, or increased. In the Huntington's type of chorea the mind is initially and preëminently dulled, and there are forced movements, and attitudes which we do not find in the family ataxias.

Multiple Sclerosis. This occurs in young people, and resembles in some particulars the type of Marie. It may also be hereditary, but in a well-marked case there are conspicuous differences. There is an intention tremor in the place of a general shakiness; the speech is staccato, instead of being slurring or elided; the limbs are spastic, instead of being ataxic, and there is an absence of swaying on attempting to stand erect. It is said by some that the nystagmus of multiple sclerosis is in all directions, while that of hereditary ataxia is lateral only.

TREATMENT. Prophylactic treatment must be confined to forbidding the mother with such a strain in her blood to nurse her children. They should be brought up with the utmost care during the period of puberty and early adult life. If we have a developed case, hygiene should be as careful as circumstances would allow. Some method of spinal extension might be worth a trial with a view to increasing the circulation in the cord. Tonics and nerve stimulants are of little value.. If a period of stasis is reached reëducation of the muscles, as in tabes, might be tried.

It seems as though Nux moschata, Alumina, or Phosphorus might be worth a trial, but in the author's limited experience in this rare disease neither his attempts, nor those of the regular school produced any effect, though Dana reports a case of Stilwell's which was benefitted by the use of antipyrin.

HEREDITARY SPASTIC PARAPLEGIA.

DEFINITION. This is an extremely rare form of spastic paraplegia, which has been found in several members of the same family, more commonly in the male members, and has been traced out for eight generations in a case of Spiller's. If it skips a generation it seems to disappear. (Bagley series.)

AGE. It has appeared as early as the first year, and as late as the fifty-sixth.

ETIOLOGY. It is evidently a developemental defect, arising from neuroses, insanity, syphilis, alcoholism, or consanguinity of the parents, and often excited in the parents by some one of the infectious diseases.

PATHOLOGY. The changes are scleroses of the lateral tracts, especially the crossed pyramids, but changes may be present in the bulb or the brain.

SYMPTOMATOLOGY. The symptoms are those of a spastic paraplegia. As years go on contractures become pronounced, and the rigidity of the legs and the deformity of the feet, frequently talipes equinus varus, make walking impossible. The upper extremities are rarely involved in the pure form. As was said of primary spastic paraplegia, these cases at times involve the upper extremities, and there are intention tremor, staccato speech, nystagmus, and optic atrophy, all of which form the symptom-complex of multiple sclerosis. Intelligence invariably is intact.

PROGNOSIS. It may be a progressive disease, leading to complete disability, or halt at any stage.

TREATMENT. The only treatment which promises large result is the section of the posterior roots from the cervical or lumbar plexuses (method of Foester), or the isolation of the nerves to the morbid muscles by the injection of alcohol (Schwab and Allison), or the severing of the whole, or part of the nerve only (Stoffel). The after massage and exercise of the antagonists is essential. If the disease shows latency, massage may restore some muscular groups; for in some localized losses with a similar pathology, clinical recoveries have been seen.

THERAPEUTICS. Phosphorus, Phosphoric acid, Baryta carb., and remedies affecting sclerotic deposits are worth a trial.

CHAPTER VII.

THE PROGRESSIVE MUSCULAR ATROPHIES AND DYSTROPHIES.

DEFINITION. These are all diseases, which with some initial difference finally terminate in selective atrophy, which precedes, and is the cause of a paralysis of variable degree and extent. While many types have been isolated, they are liable to fade into one another, and lose distinctiveness.

While describing the atrophies and dystrophies, it seems well to consider a disease where, as yet, we can discover no tangible changes in either nerve or muscle, but in which clinical observation would lead us to believe that there had been some sort of change in the muscle-elements. This disease is Myasthenia gravis or Asthenic Bulbar Palsy.

PROGRESSIVE MUSCULAR ATROPHY.

SYMPTOMS OF TYPICAL CASES. Progressive Muscular Atrophies. There is the gradual wasting of a part of a limb, or several parts of the body, producing a paralysis, without sensory changes, and without an initial fever, or any general disease to explain its presence.

A Progressive Muscular Dystrophy, called also a Myopathy, differs only in its earlier phenomena. There is a pseudo-hypertrophy in the muscles, with a progressive weakness before the atrophy, which finally becomes as pronounced as if the atrophy were the initial symptom.

AGE, SEX, and HEREDITY differ so widely in their influence upon the developement of the different forms that they will be referred to as the separate divisions of this disease are described.

ETIOLOGY. No cause can certainly be proved, but it is probable that a fault in embryonic developement lies at the root of many, and perhaps of all cases. On this as a basis, exposure to cold, deprivation of food, the intoxications, and infections, and emotional shocks, or physical injuries, are any one of them sufficient to produce the condition. Syphilis has been a cause (Abrahamson), and it has been a late result of anterior poliomyelitis and of tabes. In some cases the degeneration alone is evident, while occasionally an extensive endarteritis is the basic pathological change.

PATHOLOGY. Progressive Muscular Atrophy is caused by a simple shrinkage of the lower motor neuron. That includes cells, dendrites, and axons alike. There is no change, such as we find in the ordinary degenerations, but, preserving its usual form, the axon becomes too small for its lymphatic

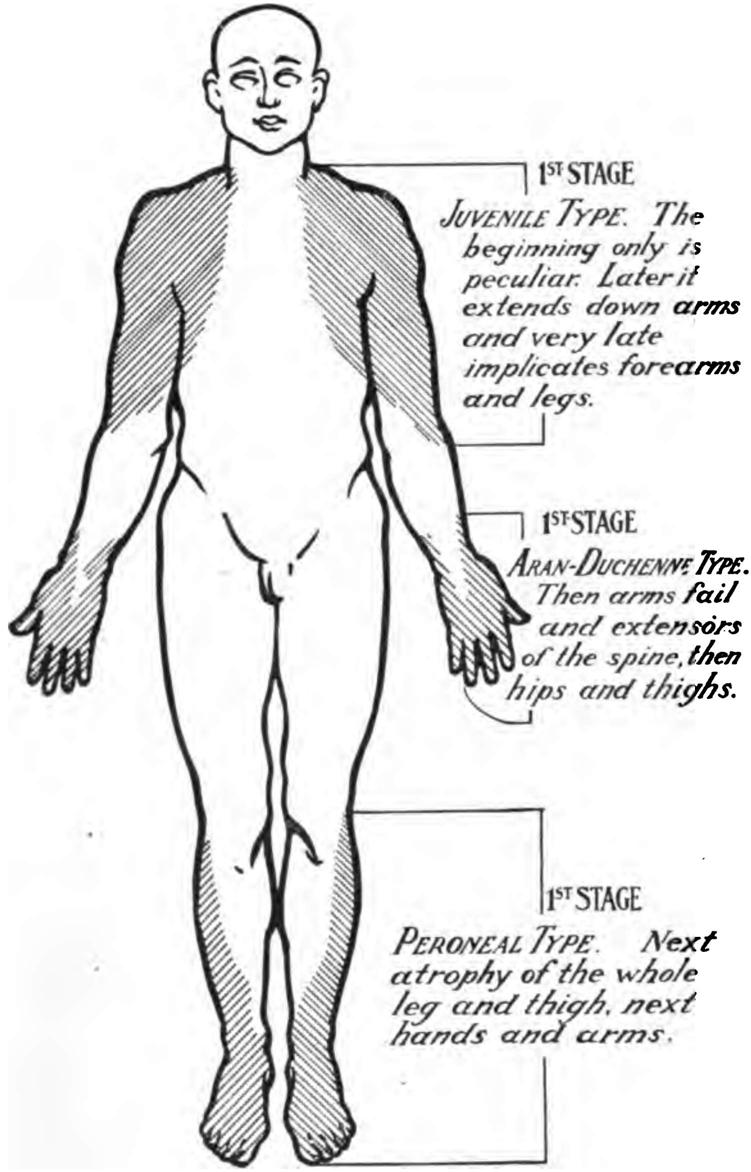

1ST STAGE
JUVENILE TYPE. The beginning only is peculiar. Later it extends down arms and very late implicates forearms and legs.
1ST STAGE
ARAN-DUCHENNE TYPE. Then arms fail and extensors of the spine, then hips and thighs.
1ST STAGE
PERONEAL TYPE. Next atrophy of the whole leg and thigh, next hands and arms.

FIGURE 68. MUSCULAR ATROPHIES.
(Modified from Mettler.)

envelope, and finally there may be only a rounded, nucleus-like body, or small mass of granules, to mark the former site of a cell. The dendrites, which formerly were collectors of nervous impulses from nerve-terminals in the neighborhood, and axons, which transmitted motor and trophic impulses to the structures to which they were distributed, also shrivel and become inert. This applies not only to the large and small motor-cells, but also to those which give rise to the association-tracts. The cells of the column of Clarke are also shriveled, and so the whole antero-lateral part of the cord is shrunken. In the muscles there is a simple atrophy of the fibres, with a loss of striation, and fatty deposits are also found. Occasionally an hypertrophied fibre will be discovered.

PROGRESSIVE MUSCULAR DYSTROPHY.

Muscular Dystrophy arises from a lack of developemental power in the muscle-fibres. In the pseudo-hypertrophic form there is initially an edema of the muscle, with an albuminous deposit, causing an enlargement. Whether or not the muscle is initially enlarged, the final result is the same. The muscular tissue loses its distinctive appearance and quality, and may be found in any stage of degeneration, even to being replaced by drops of fat. Some fibres will shrink from their ordinary size of 5 centimetres in length, and 5 to 15 millimeters in breadth, to mere threads, while others, as has been said, will altogether disappear. In the narrowed remnants, some striations may remain, but they will be farther apart than is normal, as if the weak muscular thread had been stretched in its effort to functionate. It appears to be a developemental fault, since in embryonic life there is an excess of fibres, which are probably normally removed in somewhat the fashion above described. In this disease, a normal process has been carried out to an excessive degree at too late a period.

Differential Diagnosis. This pathological statement does not comprise all the evident differences between the two conditions. Clinically, we find that a Dystrophy differs in these points from an Atrophy: (1st) Age at onset is earlier. (2d) Hereditary influence is generally traceable. If present at all in an Atrophy, it is generally through the paternal line, while in a Dystrophy, where it is usual, it comes through the maternal line. (3d) The point of initial attack is different, since in a Dystrophy the hands do not suffer first, but, on the contrary, the large muscles of the arm, the shoulder, or of the pelvic girdle. (4th) Bulbar paralysis does not occur. (5th) Muscles do not waste in groups, conforming to the localization of their nervous supply in the segments of the cord. (6th) Apparent hypertrophy of some muscles may co-exist with atrophy of others, or there may be a congenital absence of some muscles. (7th) Fibrillary twitchings are, as a rule, absent. (8th) There is never a typical reaction of degeneration, but, as muscles waste, there is a diminution in electrical response to an equal extent to both the galvanic and faradic currents. (9th) The deep reflexes are not exaggerated, as in the deuteropathic form (amyotrophic lateral sclerosis), but if the quadriceps extensor is wasted, the knee-jerk will of course disappear. These pathological and clinical differences very definitely separate the atrophies from the dystrophies, but there are in both grand divisions symptom-complexes which are quite definite, and so differ among themselves in method and site of onset, rate, and law of extension, and effect upon future activity, and even upon life itself, that some sort of subdivision has been forced upon every writer. The following scheme has seemed to me to be the most reasonable and illuminating:

Divide the Progressive Muscular Atrophies thus:
  Chronic or Subacute Anterior Poliomyelitis.
  Atrophy of the Aran-Duchenne type.
  Atrophy of the Charcot-Marie-Tooth type.
  Hereditary type of Werdnig and Hoffman.
Divide the Dystrophies thus:
  Hypertrophic or Pseudo-hypertrophic type.
  Erb's Juvenile type.
  Facio-scapulo-humeral, or the type of Landouzy-Dejerine.
  Hereditary form.

DIAGNOSTIC FEATURES OF THE VARIOUS TYPES OF ATROPHIES AND DYSTROPHIES.

AGE: INFANCY. Facio-scapulo-humeral. 2 to 4, or at any rate in early childhood.
  Hereditary form of Atrophy. Before 2d year. (6 to 12 months—Ormerod.)
  Pseudo-hypertrophic Paralysis. 2d to 20th year.
CHILDHOOD. Erb's Juvenile type of Dystrophy. 12 to 16 or 20.
  Hereditary form of Dystrophy. 8th to 10th year.
EARLY LIFE. Charcot-Marie-Tooth type. Before 20.
ADULT. Aran-Duchenne type of Atrophy. Rarely before 20; generally 25 to 45.
LATE ADULT. Chronic, or Subacute Anterior Poliomyelitis.
LOCALIZATION. (This indicates the site of initial and usual lesions.)
  Facio-scapulo-humeral type of Dystrophy. Face, shoulder, arm. Later it may involve back and legs, and rarely, all parts.

Charcot-Marie-Tooth type. Calf, foot, leg, and later the arms.

Aran-Duchenne. Hand, arm, shoulder, back, (lower limbs, late, and very rarely, if ever).

Chronic or Subacute Anterior Poliomyelitis. All four extremities (neck and back late, if ever).

Hereditary form of Dystrophy. Back and lower extremities.

Hereditary form of Atrophy. Back and thighs, arms, legs, and hands.

Pseudo-muscular Hypertrophy. Calves, legs, back; later shoulders, arms (hands always free).

Erb's Juvenile type of Dystrophy. Shoulder, arm, back, leg, calf (hands free).

FIBRILLATION. All Atrophies.

REACTION OF DEGENERATION. All Atrophies.

HYPERTROPHY OR PSEUDO-HYPERTROPHY. Pseudo-hypertrophic paralysis. Erb's Dystrophy.

HEREDITARY. All Dystrophies, and Charcot-Marie-Tooth type of neural Atrophy. Dystrophies through the maternal line; when an Atrophy is hereditary it descends through the paternal line.

CURABLE. Chronic and Subacute Anterior Poliomyelitis, sometimes.

FATAL. Chronic and Subacute Anterior Poliomyelitis, sometimes. Hereditary type of Atrophy before or by the 6th year.

COURSE. Rapid. Chronic or Subacute Anterior Poliomyelitis. Hereditary type of Atrophy does not last over 4 years.

Slow. All other Atrophies and Dystrophies.

CHRONIC OR SUBACUTE ANTERIOR POLIOMYELITIS.

(*Atrophy of the Arm and Leg Type.*)

DEFINITION. An atrophy of a late and rare form affecting the arm or leg, and finally all the extremities.

THE SYMPTOMS OF A TYPICAL CASE are these: A person in late adult life developes an atrophy in a muscle, or in a member, which spreads over all four extremities. This process spares the trunk, diminishes the deep reflexes, displays the reaction of degeneration, but does not disturb the sphincters, or sensation, and while it may be fatal to life is sometimes curable.

AGE. Late adult life.

SEX. Sexes about equally. Not familial.

ETIOLOGY. Doubtful, but probably some form of toxemia. Has developed in diabetes; Erb says from traumatism; Gowers says intemperance, venereal excesses, cold, and falls on the back.

SYMPTOMATOLOGY. While a whole limb may be attacked, it may seize upon single muscles here and there, since muscles tend to become involved as different contiguous cell-groups become, in turn, the seat of the peculiar degeneration. The atrophy tends finally to involve the four extremities and in some cases the trunk and the bowels are also the seat of atrophy. The deep reflexes are diminished, but the sphincters are spared, and there is no diminution of sexual power. The atrophy is preceded by fibrillation. Electrical changes may precede the atrophy, and while the complete reaction of degeneration is to be expected, the preservation of some fibres of a muscle may render it only partial in some localities. The only sensory changes are occasional pains simulating rheumatism.

COURSE. Invasion period is from two weeks to some months, and then the disease may halt, or show a gradual extension.

PROGNOSIS. The slower the invasion the slower the subsequent progress. Cases arising from traumatism are most likely to reach their full developement in six or eight months, and then may rest at that stage of debility, or slowly improve to some extent. Debility may be stayed at any point, or the person go on to complete helplessness, but with no tendency to death, or may die from pneumonia, or cardiac debility.

ARAN-DUCHENNE TYPE.

(*Shoulder-arm-body type.*)

DEFINITION. This is the usual form of a muscular atrophy, picking out the muscles in a selective fashion, and producing a very extreme grade of disability. It is very apt to invade other parts of the cord besides the cells of the anterior horn, and adds some symptoms of the involvement of the upper neuron. Gowers says, that he has never seen it pure in type.

THE SYMPTOMS OF A TYPICAL CASE. A man in the prime of life developes very slowly an atrophy in the muscles of the shoulder, then the arm, and possibly of the body, which spares the legs. There is some degree of reaction of degeneration. It is incurable; it may remit, and may be fatal.

AGE. Generally from twenty-five to forty-five.

SEX. More males.

HEREDITY. Not a factor.

ETIOLOGY. Not always known, but may follow traumatism (I have seen it follow a blow on the vertebra prominens), infections, intoxications, and exposure, and sometimes complicates tabes dorsalis.

PATHOLOGY. This is peculiar only in its localization. The cell-shrinkage is at first limited to the 1st dorsal, 8th and 7th cervical. These segments furnish the nerve-supply to the hands. After these have become severely affected, it next attacks the 5th cervical, which supplies the deltoid, and then the adjacent cell-groups, which go to the biceps, coraco-brachialis, and supinator longus. It finally involves all the cell-groups of the cervical enlargement, and then attacks the groups in the lumbar enlargement. It may become general.

SYMPTOMATOLOGY. Some rheumatoid pains may usher in the disease, and there may be a feeling of superficial numbness, or the very first symptom may be a wasted appearance of the ball of the thumb (thenar eminence), or of the thickened muscular mass below and to the outside of the little finger (hypothenar eminence). This allows the thumb to drop back to the plane of the fingers, and the result is "Ape Hand," and as the lumbricales, and the interossii become wasted, and the articulations relaxed, the distal extremity of the first phalanges is retracted, and the result is the "Claw," or "Preacher's" Hand. Next, the deltoid is wasted, and the arm drops at the shoulder. The other muscles of the shoulder-group soon follow, and the shoulder-blades swing down and out, giving rise to the very characteristic "Angel-wing" deformity. From the atrophy of the biceps, and coraco-brachialis, the power of flexing the forearm upon the arm is lost, and the member swings loosely, often partially dislocated. The point of the shoulder is allowed to drop from atrophy of the trapezius, and serratus magnus. The wasting of the levator anguli scapulæ, inserted into the posterior border of the scapula, allows it to drop, and the lower angle to stand out—"Angel-wing." The same process next affects the muscles of the trunk and back. The legs are usually spared, but are finally as helpless as the arms, if the disease manifests its fullest possibilities. In all forms of progressive muscular atrophy, it is to be understood that paralysis follows and is proportionate to the atrophy. The disease may rise to the nuclei of the bulb. The pupils may be stiff and unequal. In these cases there is possibly a syphilitic etiology, or it may be a complication of tabes. Fibrillation precedes the atrophy. Reaction of degeneration is present to the degree of the completeness of the muscular loss. The extremities are livid and cold.

COURSE. The progress is so gradual, that it may be many months before one can be certain of the diagnosis, and then it may be two years more before the shoulders are affected, and several years more before the full degree of disablement is attained. The steps in the process may be marked by long remissions.

PROGNOSIS. Recovery is impossible, but the disease may stop at any point, and after it is fully developed the losses may be minimized by some of the unaffected muscles taking on new functions. If the bulb is implicated, death is the result.

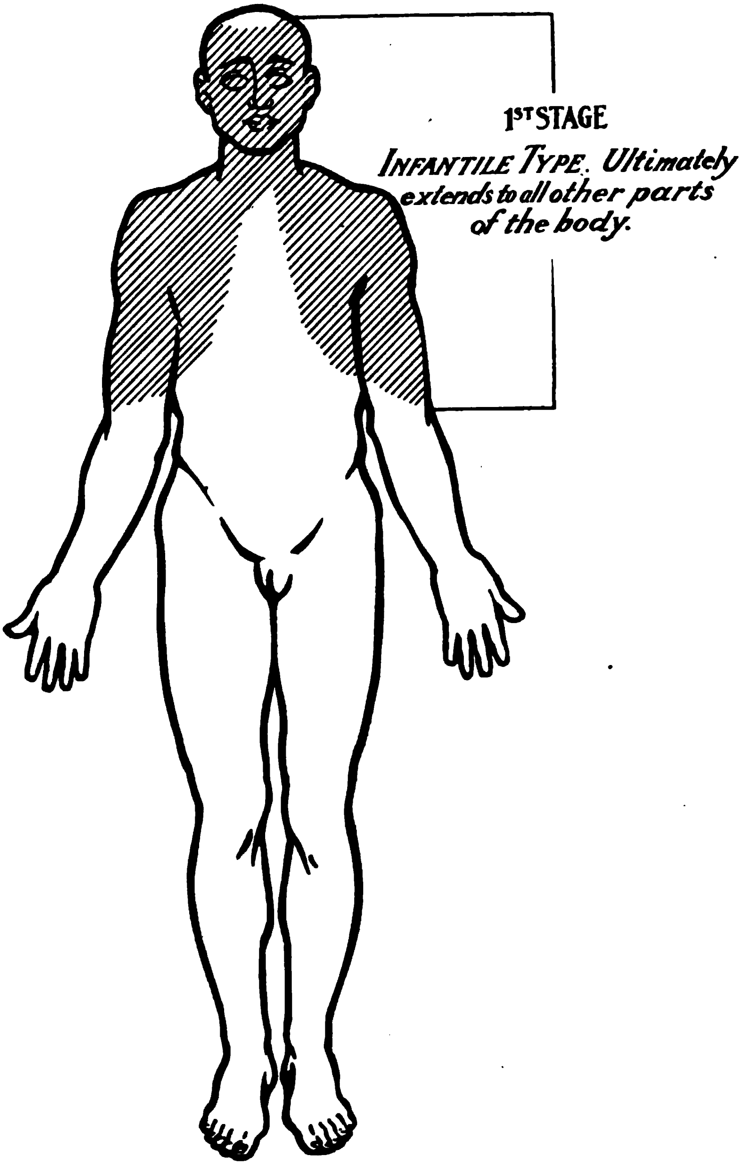

1ST STAGE
INFANTILE TYPE. Ultimately extends to all other parts of the body.

FIGURE 69. MUSCULAR ATROPHIES.
(Modified from Mettler.)

Familial Type of Werdnig and Hoffman.

Definition. This is a rare form of muscular atrophy which is familial, widespread, and fatal. The invasion is subacute, or chronic.

Symptoms of a Typical Case. An infant or young child, who is very fat, becomes weak in the hips and back, next in the arms, and then in the legs. Atrophy follows the weakness, and the bulbar muscles may become affected. It is fatal before the sixth year.

Age. It is usually said to begin between the sixth and the twenty-fourth month of life, but Abrahamson (who recognizes three types) states that it may begin in the first days, or the first month of life.

Pathology. There is a congenital fault plus an intoxication of some sort. The lower motor neuron may be affected in whole, or in part, and in addition the muscular tissues are embryonic.

Symptomatology. The affected children are very fat, and they may be attacked shortly after birth, or after they begin to walk. The atrophy of the muscles is at first masked by the subcutaneous fat, as in the pseudohypertrophic form of dystrophy, so that the weakness of the hip, back, arms, legs, and finally of the hands seems to antedate it. It is a flaccid paralysis to complete helplessness, with diminished deep reflexes, and the reaction of degeneration. Bulbar symptoms have been observed. The mind is unaffected, and sensation is unimpaired.

Prognosis. Death occurs before the sixth year.

Progressive Neural Type—Myostic Type—Charcot-Marie-Tooth Type.

(*Peroneal Type.*)

Definition. An intermediate form between the atrophies and the dystrophies, since at times the nerves, while at others the muscles, are the point of initial attack. A simultaneous degeneration is, however, most common. It is called "neural," from the fact, that from the spinal-cell to the muscle-cell, all parts of the lower motor-neuron are affected. It was called by others "myositic," since the initial change might be in the muscle instead of the nerve, and by all it is called "peroneal," since the initial weakness is always in the peroneal group of muscles.

The Symptoms of a Typical Case. A person between the tenth and the fortieth years shows an atrophy in the peronii, and acquires double talipes varus. After a long remission the whole leg atrophies, including the foot, and then the muscles of the thigh. Next atrophy attacks the hands and arms, finally leaving all four extremities cone-shaped. Electrical reaction is depressed, as are the deep reflexes; sensation is only slightly disturbed. Not fatal, but incurable.

AGE. The second half of childhood, and the latest limit of its appearance is placed from the thirtieth to the fortieth year.

SEX. Shows no difference.

HEREDITY. It descends through the paternal line, although it may skip a generation, which classes it with the atrophies. It has been seen in several members of a family (Sherrington has reported twenty-six cases in one family), in which it resembles a dystrophy.

ETIOLOGY. It has been attributed to measles, and other infections, but it is certainly not syphilitic.

SYMPTOMATOLOGY. The muscles on the outside of the leg begin to waste, and gradually double clubfoot results, which may be the only condition produced for several years. The remission has been so complete at this point, that tenotomies have been performed for what appeared to be a completed process. The anterior, and then the posterior tibial muscles are wasted, and next, after years perhaps, the vastus externus, and then the adductors are also involved. After the tibials, the intrinsic muscles of the foot are atrophied. Now, after another halt, the hands, and then the arms atrophy, and from the unequal losses "Claw-hand" results. The disease may stop at this point, or may go on to a general atrophy. The atrophy is so selective, and follows such a definite order, that the limbs assume a cone-shape both in the legs and arms. Fibrillation and a fine tremor may be present, and a lowered electrical excitability, and often an early, but incomplete, reaction of degeneration. Sensory disturbances are limited to a slight degree of numbness. Reflexes are decreased, and knee-jerk may be abolished.

COURSE. The progress is, as has been said, very slow, and years may be consumed in the process. It may stop at any point, and long intermissions are the rule.

PROGNOSIS. It is not apt to imperil life, as no vital centers are involved. The losses are apt to be permanent.

HYPERTROPHIC OR PSEUDO-HYPERTROPHIC PARALYSIS.

DEFINITION. This is a form of dystrophy, in which there is a hypertrophy, in some of the muscles at least, before the final atrophy.

THE SYMPTOMS OF A TYPICAL CASE. A young child seems over-developed in the legs, then becomes weak, and this may apply to all muscles, even to those of the tongue. The muscles of the back grow weak before those of the shoulder. Atrophy now ensues, and hypertrophy will be present in one part, with atrophy in progress in another. No reaction of degeneration, or reduction of deep reflexes appears until atrophy occurs. Progress is slow, attempts at cure futile, fatality not usual.

AGE. It may occur from the earliest to the latest periods of life, but is most frequent from the second to the twentieth years.

SEX. More males than females.

HEREDITY. It may be transmitted from one generation to another, through the mother, (atrophies through the father), and may also be familial, i. e., several members of a family being affected.

SYMPTOMATOLOGY. The change which first attracts attention is an enlargement of the muscles of both the thigh and the calf. It is at first considered a sign of perfection of developement, but soon the child is found to fall easily, and to have little endurance, and the muscles, while enlarged, are found to be very weak. The tongue may also be the site of hypertrophy. At the same time, perhaps later, examination of the upper limbs will disclose an atrophy, and however distributed, the peculiarity of the disease rests upon an hypertrophy preceding a subsequent atrophy, and, also, that while one part is hypertrophic, another is becoming atrophic. The erectors of the spine are weakened at an early date, and before the scapular muscles show an atrophy, a line dropped from the cervical spine will fall clear of the buttocks, on account of the pushing back of the shoulders in the attempt to keep erect by posture, rather than, as is customary, by the tonicity of the muscles of the spine. The hypertrophy is not absolutely confined to the muscles of the lower limbs, but is most certainly, and most prominently there. The general rule is this: hypertrophy is in the glutei, calf-muscles especially, and sartorius in the lower limbs, and in the deltoid, infra-spinatus, and triceps in the upper. Atrophy is in the pectoralis major, trapezius, serratus anticus major, latissimus dorsi, biceps, rhomboids, brachialis anticus, and later, in the quadriceps femoris, adductors, and then in the previously hypertrophied muscles of the lower limbs. The orbicularis oris, and palpebrarum atrophy (Starr says these are spared), and a mask-face may result in a late stage of advanced cases. The wasting does not affect the two limbs symmetrically, and the deltoid and the spinati do not waste along with the biceps and supinator longus, as in the atrophies. If the trapezius wastes, it is affected as a whole, and the upper part is not spared, as is the case in the spinal palsies.

The peculiarity of the atrophy produces peculiar deformities. The point of the shoulder drops from the atrophy of the trapezius, and serratus magnus, and the deltoid, and the scapula is loosened, and swings out from the wasting of the rhomboids, serrati, and levator anguli scapuli. Since the erectores-spinæ are weakened, the spine falls forward when the patient is in a sitting posture (lordosis), and its loss embarrasses the efforts to stand erect, or to rise.

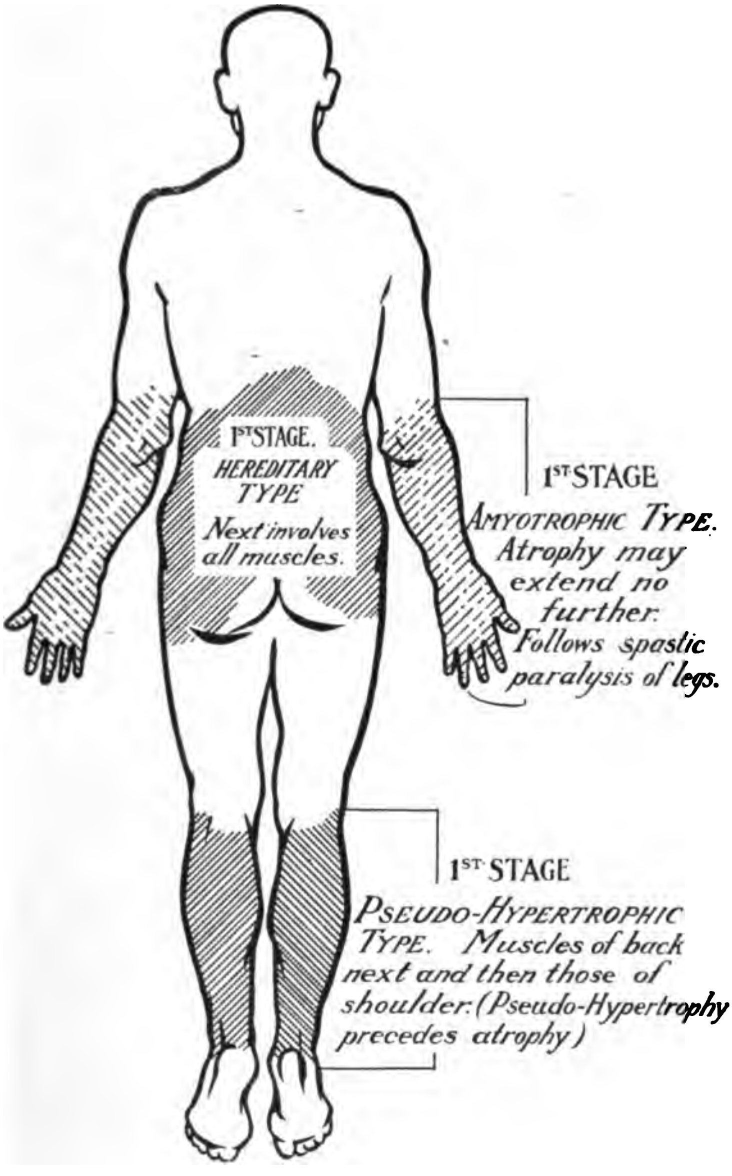

1ST STAGE.
HEREDITARY TYPE
Next involves all muscles.
1ST STAGE
AMYOTROPHIC TYPE.
Atrophy may extend no further.
Follows spastic paralysis of legs.
1ST STAGE
PSEUDO-HYPERTROPHIC TYPE. Muscles of back next and then those of shoulder. (Pseudo-Hypertrophy precedes atrophy)

FIGURE 70. MUSCULAR ATROPHIES AND DYSTROPHIES.
(Modified from Mettler.)

Function is impaired in many ways. The patient can hardly sit erect without support; walking is waddling, with a tilting of the pelvis, first to one, and then to the other side; stair-climbing is difficult or impossible; rising from a seat is only accomplished by pushing the body up by the arms; resting upon the knees. The typical losses are best brought out by the following test. Place the child upon its abdomen, upon the floor, and ask it to rise. It must first roll to one side, to get the knee under the body; supporting itself by this knee and the other arm, the other knee is gotten under the body; then one foot is placed upon the floor, and then the other; supporting the trunk with one hand, the other is placed upon the opposite knee, and the trunk pushed up to a vertical position, and the other hand on the knee of its side completes the process of walking the hands up the thighs, until the body is finally erect.

This wasting, and consequent paralysis may go on to any extent, but the muscles of the forearms and hands are usually spared. Contractures and deformities are common in the limbs, talipes equinus in the feet, and lateral scoliosis in the spine, from the unequal atrophy of the spinal muscles. Electrical excitability becomes reduced, as the muscular elements become unable to respond, but there is no reaction of degeneration, as in the atrophies. Fibrillation is absent. Reflexes are normal, as long as muscles have power. Sensation is not at all changed, unless the degeneration in the muscles is very rapid.

COURSE. Is so slow, as to be almost unnoticed at first, but in from four to ten years, the patient is helpless.

PROGNOSIS. The disease may not go to the extent of helplessness, but usually does, and cure is impossible. It does not tend to death, except from enfeeblement, which renders the person very vulnerable, especially to respiratory diseases.

ERB'S JUVENILE DYSTROPHY.

DEFINITION. A dystrophy which differs from the pseudo-hypertrophic type in the fact that it commonly occurs in youth, rather than in infancy, and that the initial point of attack is in the shoulder, rather than in the leg.

AGE. Early youth, twelve to sixteen, say most authors, but some extend the period to thirty-five, or even later. The majority are in the first-named period.

SEX. Equal.

HEREDITY. Not markedly hereditary, but is apt to be found in several members of a family.

SYMPTOMATOLOGY. The pectorales, trapezei, latissimus dorsi, and rhomboids are first involved, but the deltoid often escapes. Later the triceps and biceps atrophy, but the muscles of the forearm, except the supinator longus, and of the hand, escape. It may stop at this point, but if it does progress, it will next involve the muscles of the back, and then the muscles of the anterior part of the thigh, and then those of the calf, especially the peronei. In this disease, the atrophy is most often in the leg, while the initial hypertrophy is exhibited in the muscles of the upper extremity. It is selective, being mostly in the deltoid, if that is involved, and the supra-spinatus next; sometimes the sartorius and gastrocnemius in the lower limb. The result is lordosis, talipes, and paralysis as in the preceding disease. There is no fibrillation, nor reaction of degeneration.

COURSE. Progress is very slow.

PROGNOSIS. Bad for recovery, but does not commonly cause death.

FACIO-SCAPULO-HUMERAL.

(*Type of Landouzy-Dejerine.*)

DEFINITION. It is perhaps the earliest type of the disease, finally ending in muscular atrophy, and is the most complete, this disease furnishing those museum curiosities known as "living skeletons." Peculiar in the atrophy of the face.

THE SYMPTOM OF A TYPICAL CASE is an atrophy occurring first in the face, the patient being an infant, or a young child. The atrophy finally extends over the whole body, attacking the shoulder next, and then proceeding downward. The peculiarity is the age of the patient, the tapir-like mouth, and the apparent elongation of the neck, from the dropping of the clavicles. The progress is very slow and uniform, and life is not immediately endangered.

AGE. Most often begins in infancy, sometimes in childhood, and exceptionally in adult life.

HEREDITY. This is directly hereditary, familial, and through the maternal line, like the other dystrophies.

SYMPTOMATOLOGY. Weakness and wasting are first manifested in the face, and most authors say that this wasting is particularly marked in the orbicularis oris, and palpebrarum (Starr says that the eyelids escape). All agree that the muscles about the mouth, and the finer muscles of the lower face are attacked, and their function impaired. The levator menti, and the risorius are so affected that the lips cannot be apposed strongly. and the zygomaticus is also atrophied. The process extends to the shoulder, and finally over the whole musculature of the body. The rest of the process differs in order of appearance only from the other forms of dystrophy.

With the whole body weakened by the atrophic paralysis of its muscles, the distinctive feature of this disease lies in the fact that the mouth is tapir-like, the saliva dribbles, expression is lost, the cheeks puff out in attempts to talk, and it may be impossible to close the eyes completely. The neck seems to be elongated, from the dropping of the clavicles.

COURSE. Progress is so slow, and the atrophy is so uniform, that many people think that the patient is simply an instance of constitutional thinness.

PROGNOSIS. Hopeless for recovery.

TREATMENT. The cure of either a muscular atrophy or dystrophy is so far from the probability, that prevention ought to bulk largely in our view of the subject. The question of marriage and offspring is complicated by ethical and sociological considerations to such an extent that it cannot be settled by a dictum. It will be granted, however, by everyone, that the multiplication of probable invalids is not desirable, and also that the children of parents with an hereditary taint should be reared with the most scrupulous hygienic care. The child should be developed, without forcing, but at the same time without pampering. The developement of physical and mental resistance should be the aim. Traumatism should be avoided, and if suffered should be the subject of intelligent care, since hereditary instability of tissues is frequently awakened into definite disease by traumatism. It has been considered that there can be little difference in the hygienic treatment of the atrophies and dystrophies, except that we should feed fats to the atrophic, and not to the dystrophic. In the developed cases the results of treatment have been very slight, except in so far as we are able to conserve their comfort, and modify their disabilities. Massage is always beneficial, if it is not carried to exhaustion, which is easily produced. Resistive exercises also keep up the nutrition of the muscles, and the Zander type of apparatus is sometimes applicable. Most authorities advise moderate activity, and an avoidance of continuous rest for any considerable period, fearing that the patient will become bed-ridden. Oppenheim advises the most absolute rest, and probably exercise with rest periods is the most beneficial regimen. Evident deformities can be lessened by supporting gloves, high stiffened shoes, the angel-wing scapulæ can be strapped, and perhaps braces for a whole limb may be demanded.

These are merely conservation methods, endorsed in the main by all; but can anything be done curatively? If there is an evident toxemia, there are methods for antagonizing it. More generally there have been attempts to stimulate the appropriate spinal centers, by mustard, or other stimulants, to the spinal region. The Pacquelin cautery has been applied lightly every week, or every other week. Kellogg of Battle Creek states that benefit has followed the use of a hot water pour on the spine for a period of from five to eight minutes, followed by a cold pour of from fifteen to twenty seconds, or an alternation of each for fifteen seconds, or alternate cold and hot compresses to the spine, this latter for atrophies alone. The application of electricity combines with this local stimulation a direct effect upon the nutrition of the neuron, and to obtain the maximum of effect it should be carried out with careful attention to technique. King advises, in atrophy, giving a moderately strong galvanic current through the spine. Place an anode, three or four inches square, over the diseased spinal segment, and the cathode over the sternum. A current of fifteen to twenty milliamperes should be used. After a little time, the anode should be moved its length, until the whole diseased area is successively covered in a treatment lasting about ten minutes. On the muscles, use a faradic current of tension, so as to make firm contractions as the electrode is moved rapidly over them, avoiding strong clonic contractions, which tire the muscle. Take from five to ten minutes, according to the number of muscles involved. Erb says: "Pass the moistened electrode over the surface of each affected muscle, keeping the electrodes close together, and using a moderate primary current. Do not interrupt very frequently. Treat only the muscles which react to faradism, and pay most attention to the most important muscles, and terminate the sitting by attention to any muscles which are threatened with an invasion of the disease.

THERAPEUTICS. The remedies with most evidence of efficiency are Argentum nit., Phosphorus with faradism, Cuprum, Plumbum, Gelsemium, Baryta, Mercurius. Others advised are, Zinc. met., Arsenicum, Physostigma ven., Belladonna, Nux vomica, Strychnia, Lathyrus, Sulphur, and the anti-psorics.

Physiological medicine mainly relies upon Strychnia in some dosage. In the atrophies, Gowers says that by the mouth it is of no value, but the nitrate, hypodermatically, is valuable. He begins with a dose of 1-100th of a grain daily, cautiously increased to 1-5th. It has arrested very bad cases. Starr does not believe in it, but would rather give it by the mouth, 1-50th of a grain daily for four days, and then Arsenic 1-50th, the other three days of the week. Glandular extracts have not produced any beneficial effect.

BULBAR PALSY.

PROGRESSIVE BULBAR PARALYSIS—PROGRESSIVE LABIO-GLOSSO-LARYNGEAL PARALYSIS—POLIO-ENCEPHALITIS INFERIOR CHRONICA OF WERNICKE.

DEFINITION. It is a degeneration of the motor nuclei in the bulb, which give rise to the nerves which innervate the muscles of speech, of facial expression, of phonation, deglutition, and finally of respiration. This results in a slowly developing atrophic paralysis of these muscles, and the more or less complete abolition of their functions.

THE SYMPTOMS OF A TYPICAL CASE. A person in late adult life will develope a slurring speech, in which some consonants are poorly pronounced. This disability will increase, and it will be found to depend upon a paralysis, with wasting of the tongue and lips, which will become very marked in the entire lower portion of the face. Apoplectiform seizures may be an incident of the later stages of the disease.

DIFFERENTIAL DIAGNOSIS. To be differentiated from Dysarthria, Pseudobulbar Palsies, Tumors or Hemorrhages, Thrombosis or Embolism of the Bulb, and the inflammations of this region, which are termed Polioencephalitis superior or inferior, according as they are located above, or include the nucleus of the facial nerve, Bilateral Facial Palsy, Multiple Sclerosis, Amyotrophic Lateral Sclerosis, Myasthenia Gravis, and Infantile Bulbar Palsy.

FREQUENCY. It is a rare disease idiopathically, but may occur in the terminal stage of tabes, multiple sclerosis, syringomyelia, and amyotrophic lateral sclerosis.

AGE. The period of late adult life, but while very rare before forty, has been known as early as thirty, and as late as seventy.

SEX. The sexes are usually considered to be affected equally, but Dana's cases have shown a female preponderance.

HEREDITY. It is not one of the diseases in which heredity seems to be capable of demonstration, beyond a neurotic tendency.

ETIOLOGY. The actual cause is unknown, but cases have been known to follow cold, exposure, toxemias (Hoffmann quotes a case from lead poisoning), and it has seemed to have resulted from hereditary, or acquired syphilis. Overuse of the muscles of this region has been given as a cause. A case quoted was from the blowing of wind-instruments. Such a result indicates a neuropathic tendency, and stamps the underlying cause as a localized pre-senility. The exciting cause may be any form of overstrain, either general, or localized, or a reduction of resistance from infection, intoxication, or exposure.

PATHOLOGY. Speaking broadly, it is a degeneration of the nuclei in the medulla of the motor cranial nerves. Specifically the motor neurons if these nuclei are degenerated in all their parts, and the muscles to which they are distributed. There are no sensory changes, since the sensory neurons seem to resist the process. This degeneration is shown in the nuclei by the changes in the ganglion cells, and by the loss of their dendrites, and axons.

Other structures may be affected by contiguity, and therefore fibres passing to lower or higher levels may be attacked, the pyramidal fibres particularly. Palsy of the hypoglossal would paralyze the tongue, of the spinal accessory would paralyze the trapezius in the upper third, and the sterno-cleido-mastoid muscles. The degeneration of the glosso-pharyngeal would paralyze the pharynx, while the degeneration of the fibres originating in the accessory nucleus, but continuing in the pneumogastric, would paralyze the larynx. The paralysis of the pneumogastric affects respiration and heart's action. The degeneration of the facial would paralyze the superficial muscles of the face, the frontalis, platysma myoides, stylohyoid, and the posterior belly of the digastric, but since only one of the several nuclei of origin is the seat of degeneration, the loss is diagnostically limited to the muscles of the lower face. The loss of power in the lower motor part of the trigeminus, or tri-facial, would paralyze the muscles of mastication. Since the nourishment of the muscles depends upon the integrity of the cells in these same nuclei, we shall find first fibrillation of muscles, and then atrophy of them. This is analogous to any spinal degeneration involving the cells of the anterior horns, for the nuclei of motor cranial nerves are the analogue of these cells in the cord. It is like an amyotrophic lateral sclerosis, but lacks the spinal symptoms. Sometimes they are present in the arms, and hence it has been classed by some with this disease, and considered simply as a bulbar type. The nuclei are not always equally affected, and in some cases losses are erratic. Tooth and Turner found in one autopsy that there was no lesion of the 9th, 10th, or 11th, but that the motor part of the 5th, a part of the 7th, and the 12th were degenerated. In general the hypoglossal is most frequently degenerated, next the accessory part of the vagus, then the glosso-pharyngeal, then the motor part of the 5th, and lastly the facial. From this distribution we see that the functions of speech, deglutition, mastication, and facial expression are affected.

SYMPTOMATOLOGY. The clinical symptomatology demands a description, from the fact that there is a diagnostic order in the evolution of the symptoms, and because some symptoms occur, or are lacking, without apparent reason. Briefly: there are disorders of speech, then of deglutition, next of mastication, then of phonation, and at last the respiration, and the action of the heart are affected. The first symptom is paralysis of the lingual and facial muscles in succession, which condition always denotes a degeneration in the cells of origin, or of the fibres of the nerves in continuity, and this paralysis is always foreshadowed by a weakness of the muscles. It therefore follows that the patient will show fatigue only after a long conversation, and this may be an earlier diagnostic failure than any definite speech defect. Before the discovery of atrophy of the tongue, a careful examination will reveal a flickering movement in the separate fibrillæ of the muscles (fibrillation), and finally the organ will be too weak to be protruded. This preliminary fibrillation will be true of all muscles, which will afterwards develope atrophy. In speech certain consonants are produced by a modification of the current of air by the approximation of the tongue to the teeth, and are called "dental consonants;" these are d, t, and th. They are the first letters to be slurred and lost as the tongue is the earliest site of atrophy. The lips next undergo a similar change, and become weak, trembling, and atrophied. The "explosive labials" p and b depend upon the strong apposition of the lips against the air-currents, and they, therefore, are next to become imperfect, although the "aspirate labial" f, and the "resonant labial" m can still be pronounced. The palatal muscles next become involved, and it is to be noted that a slight bilateral loss is far more disabling than a much more marked unilateral palsy. The "guttural consonants" k, g, and ch are pronounced by the apposition of the base of the tongue to the soft palate. When the tongue has become weak, and the palatal muscles are also inadequate, the pronunciation of these letters becomes more and more imperfect. In this condition some of the air leaks past into the nose, and the reduction of the mouth-pressure reduces the "explosive labials" p and b to v, or f, or even m. The vowels almost alone are left to speech, but of these o and u are often imperfect, as they demand the firm apposition of the lips. This combination of losses produces what is known as Bulbar Speech, an enunciation which is blurred, nasal, and run together, in the attempt to complete a sentence before being overtaken by exhaustion. Such a condition is Dysarthria (Beevor). For the same reason liquids, and sometimes particles of food will escape through the nose. At this time we are apt to hear complaints of the only variety of sensory symptoms present, viz., there will be some degree of pain in the back of the neck, or an occipital headache.

By this time the loss of power in the facial muscles has become so pronounced that the play of expression in the *lower* face has been impaired, and in time is wholly lost. As the lips become feebler, the saliva drips from the corners of the mouth, both from the weakness of the lips, and also from the irritation of the chords tympani of the facial, which stimulates the submaxillary glands. Without apparent reason the ocular muscles are very rarely implicated, and the zygomaticus, the elevator of the upper lip, and the frontalis are exempted.

At the same time that the palatal muscles show enfeeblement, the pharyngeal muscles begin to weaken, and there is difficulty in deglutition. At about the same period, the muscles of mastication, which are innervated by the motor branch, coming down with the third branch of the 5th, show degeneration, and chewing is performed imperfectly, and with difficultv. Particles of food are apt to lodge between the cheeks and the teeth. Taste may be affected. When at length the larynx weakens in its turn, the vocal cords become paralyzed, and the voice becomes hoarse and toneless, and finally fails for connected speech. It is a degree of Aphonia, but total Mutism is not a characteristic of this disease. The lips being so weakened, the mouth is likely to remain open, giving a stupid expression to the face.

Lastly, degeneration of the nucleus of the pneumogastric causes respiration to become feeble, and this combined with the inefficiency of the larynx, produces spasms, and wheezing on inspiration. From the same nuclear degeneration the heart's action becomes irregular, and is accelerated, the pulse rate being from 120 to 140. The patient is now apt to inspire the mucus which is so freely secreted, not only producing choking, but, in addition, it is apt to produce asphyxia, or an inspiration pneumonia, which is a common cause of death.

Atrophy after a time becomes very pronounced, especially in the muscular structure of the tongue. This ensues to such a degree that the mucous membrane lies in folds upon the tongue, and the whole organ becomes thin and colorless, and lies quivering upon the floor of the mouth. In some cases, instead of emaciation, it is the seat of fatty deposit. When the contiguous pyramidal fibres are also degenerated, we shall find some amount of spastic paralysis in the arms, and less often in the legs, and under such conditions, and then only, we shall be able to elicit a jaw and malar jerk. So long as there is any muscular tissue there will be some electrical reaction, but faradism first fails, and next there is noticeable slowness in reaction to the galvanic current, and next the anodal closure will equal, or even surpass in effect the cathodal closure. This is the reaction of degeneration. Finally the muscles fail to react to either current. Late in the disease we may see convulsions of an epileptiform type followed by mental confusion, but the mind is usually clear. The patients are apt to be sorrowful, and the emotions are easily excited. The sorrow is probably from the fact that they are mentally acute enough to realize the seriousness of their condition, but such a mental state always follows lesions affecting the pons. Some writers on the contrary insist that the patients are actually good-humored, in spite of the disease, although all agree that they are emotional. There is a general impairment of health, and patients show a progressive general emaciation, due to a lack of appetite inseparable from their invalided condition, and to inability to masticate and swallow sufficient food. The pharyngeal and laryngeal reflexes are generally lost, but not invariably. The deep reflexes are usually exaggerated.

COMPLICATIONS. The disease is associated at times with some degree of progressive muscular atrophy, and amyotrophic lateral sclerosis.

There is a form which has been termed acute apoplectic polio-encephalitis inferior acuta, which arises from hemorrhage, softening after thrombi, more rarely after emboli of the vertebral, and basilar arteries. It may be simultaneous with similar conditions in the brain.

COURSE. The disease is insidious in onset, slow in developement, for a time making no progress, but remissions are rare and short, and there is never any conspicuous recession. In a few cases the onset has been acute, but only in an unusually rapid evolution of the speech defect, for even in such cases the remaining symptoms evolve at the usual rate.

PROGNOSIS. The disease rulably terminates in death in a period of one to three years, although some cases have been slower, while others have been fatal in the first year. The cause of death is either inanition, pneumonia from inspiration of mucus or food into the trachea, or it arises from some intercurrent disease to which such individuals can offer very slight resistance.

DIAGNOSIS. When the case has come to an advanced stage there is no particular difficulty in the diagnosis, but if the disease picture is still incomplete, there may be reason for doubt. In the early stage, however, no matter how inadequate the history, certain changes are sure to be present which strongly suggest the true nature of the malady. There is even then some grade of paralysis of the tongue, and it is quite diagnostic that it is bilateral and progressive, and that invariably there is some degree of paralysis of the lower face and lips accompanying it. The patient cannot whistle, even if the anterior nares have been previously closed, nor can he blow out a candle. He cannot elevate the tip of the tongue, nor protrude it, nor place it in the sides of the cheeks on request. If the finger and thumb are placed between the lips their separation is effected with little force. Even when the reaction of degeneration has begun, some response will remain so long as any muscular tissue persists. Faradism first fails, then comes the reaction of degeneration, and then all response is finally lost. Slowness of response is seen in the early stages. Sensation is unimpaired. Patients are in later middle life. Slowness of onset is a diagnostic sign, and of great importance. Of prime diagnostic importance is the *order* of invasion, and that the paralyses shall be bilateral, and marked by atrophies, and the reaction of degeneration, and shall be only such as could be predicted by the degeneration of the nuclei of the motor cranial nerves.

DIFFERENTIAL DIAGNOSIS. Dysarthria from other causes must also be excluded. If it is due to an anatomical fault in the vocal mechanism, either congenital or acquired, it will be revealed by a careful examination. If it is the result of the action of some corrosive poison it should not be beyond discovery. If it is the late result of diphtheria, tonsillar inflammation, or some other infection, there is apt to be a history of the causative disease. If this is not discoverable, its localization, the associated paralyses, or the lack of them, the rate and manner of onset, the electrical reactions, the presence, or absence of atrophies, and the age of the patient are all means of differentiation. A unilateral paralysis of the vocal cords resulting from a lesion of the recurrent laryngeal nerve makes the voice hoarse, but does not interfere with the articulation of consonants.

Progressive Bulbar Palsy is a chronic encephalitis inferior. There are a number of conditions which simulate it in respect to the existence of a similar speech-defect. These conditions are all comprised under the name of pseudo-bulbar palsies, but have a wide variety in etiology. There are two forms of polioencephalitis, which are termed the one superior and the other inferior. The first is infantile, the result of porencephaly, and is very confusing, since its progress is so slow that it mimics the adult form. The infantile form, however, is an atrophy of the bulbar nuclei and not a degeneration, and it differs clinically in the fact that it attacks the upper face, and produces ptosis, and does not affect deglutition, which is the exact opposite to the symptomatology of the adult form. The affected children also show by the tenderness of the bones of the face that they are degenerates, and they are usually the product of consanguineous marriages, (Church.) There is a second form of polioencephalitis, i. e., inferior which is the extension of an encephalitis, often as a complication of the infectious diseases, sometimes one of the points of attack of a disseminated myelitis, may be a complication of anterior poliomyelitis acuta, sometimes the sole localization of an infection. It is particularly infantile, and as it is an inflammation, and not a degeneration, its rate of onset is diagnostic, as is the fact that it is not always fatal. It attacks the nuclei in the medulla, and, therefore, its symptoms are confusing. The same is true of the polioencephalitis, superior and inferior of Wernicke, the superior affecting the nuclei above the sixth, and the inferior those below it. Repeated apoplexies may finally produce a bilateral paralysis of the tongue, hypoglossal nerve, and of the lower face, but diagnostically it is unilateral, and not bilateral at the start, and it is distinctively not nuclear, i. e., there is no atrophy of the muscles, the lingual muscles will react to faradism, and there is never a loss of the pharyngeal and laryngeal reflexes. While in progressive bulbar Palsy there may arise some degree of spasticity in the upper limbs, less often in the lower, there is a distinct double hemiplegia in the pseudo-bulbar palsy from apoplexies. Hemorrhagic lesions in the brain below the cortex are almost always in the internal capsule, and are almost certain to affect the ganglia in some degree. To lesions of these ganglia are due the emotional outbursts of tears and laughter which are quite characteristic of the psuedo-bulbar type.

Tumor usually will develop bulbar symptoms very gradually, but sometimes quite abruptly. In any case, however, the invasion will be unilateral: a long time before they become bilateral, the general symptoms of tumor will be evident.

Hemorrhages, Thrombi, and less often Emboli of the vertebral and basilar arteries, produce a suggestive symptom-complex. Hemorrhage in this locality is almost certainly fatal if bilateral, and if unilateral there could be no similarity. The suddenness of the onset, even if there was no loss of consciousness, would also differentiate it. Softening from occlusion of the vertebral or basilar arteries, or their branches, as is more usual, lacks the diagnostic abruptness of hemorrhage, nor is there unconsciousness, but the evolution of symptoms is a matter of days instead of months, as in the progressive type. While some of the cranial nerve-nuclei are sure to be involved, the distribution will be erratic, and not exclusively motor. Moreover, the accessory symptoms are peculiarly diagnostic. The patient, usually without unconsciousness, suddenly becomes helpless in the lower limbs, with paralysis of the vocal cords on one side, and ataxia upon the same side of the body as the softening. The same side of the face, and the opposite side of the body show an increased sensitiveness to pain and heat. In other words: there is a paralysis of the parts of the body spared in bulbar palsy, and in addition a crossed hemianalgesia (loss of pain perception).

Bilateral Facial Palsy might suggest bulbar palsy, but the resemblance is superficial, since the upper face is affected, deglutition and phonation are not affected, and it is almost always a phase of multiple neuritis, and hence the condition of the limbs is diagnostic.

Multiple Sclerosis. This has been a confusing possibility at times, since both diseases have a speech defect, the diagnostic differences between the two varieties being less easy of detection than is usually stated.. In passing it is best to remind the reader that later investigation has shown that cases of multiple sclerosis are seen without the speech defect which has been esteemed diagnostic. The typical defect is that the patient speaks in a staccato manner, explosively, because the form of paralysis affecting these muscles is marked by a rigidity. In bulbar palsy, on the other hand, the fault is a slurring, a clumsiness, as well as a weakness of articulation. In multiple sclerosis the facial palsy is not a bilateral weakness, but a stiffness with weakness, and is unilateral. The extremities also suffer from a stiff weakness which is bilateral, as they rarely may in some forms of bulbar palsy, but even then the sufferer from bulbar palsy does not display the intention tremor, nor the nystagmus of multiple sclerosis.

Amyotrophic Lateral Sclerosis. If to the bulbar symptoms are added the symptoms of a degeneration of the cells of the anterior horns of the cord (shown by a spastic paralysis of the lower limbs, with wasting, fibrillation, the reaction of degeneration, without sensory disturbances), we are dealing with a more general disease of the same type as bulbar paralysis, but designated by another name for purposes of differentiation only. In this case, and not in the typical bulbar palsy, the wasting and paralysis of the face, mouth, and throat will be preceded by a spastic stage, and the jaw and malar-jerk will be found.

Mysthenia Gravis. Here we find early ptosis, paralysis of the orbicularis palpebrarum (the paralyses of bulbar palsy are confined to the lower face), as well as of the other muscles from the bulbar nuclei, weakness of the muscles of the neck, so that the head rolls about, which is late if ever in bulbar palsy. There is greater and more prompt muscular weakness in myasthenia gravis, and the knee-jerks are never exalted, but show the peculiarity of ready exhaustion on repeated exhibition, as do all of the other muscles affected. There is no reaction of degeneration, but instead this diagnostic feature, viz., the response to all forms of galvanism diminishes progressively as it is repeated, but after a period of rest is found to be as energetic as at first. Remissions and recessions are frequent, and cure is not unknown.

TREATMENT. This falls into the two divisions of adjuvant and hygienic, and therapeutic. The hygiene should be of the most perfect kind, and over-exertion, particularly of the vocal organs, should be strenuously forbidden. The food should be of the most nourishing kind, there should be plenty of fresh air, and the nutritional effect of massage, both general and local. Hot baths and packs, followed by the cold douche or spray, are of use in toning up the general circulation, and any shock should be antidoted by vigorous rubbing after the bath. Massage locally may relieve some symptoms. When in the process of the disease swallowing has become difficult, great care should be exercised to prevent the abundant mucus from falling down the trachea, and causing choking, and pneumonia. Atropine is the best means of relieving this condition. There should also be at hand a number of long swabs of absorbent cotton which should be used in a routine manner, in preventing great accumulations of mucus in the mouth, and all attendants should be sufficiently trained to clean out the pharynx on a moment's notice. When the failing power of deglutition has begun to affect nutrition, the stomach-tube, or preferably the nasal-tube, should be employed, and each feeding should be followed by four ounces of water. Tracheotomy may be necessary. When the stomach has become intolerant, feeding must be by rectum, and nutritive enemata should be followed by six ounces of normal salt solution to combat thirst.

Electrical Treatment. This should be tried for a short time, but not if the case comes into one's hands with a well-marked reaction of degeneration, or if this comes on in the course of the disease. In no case should the treatment extend over ten minutes in all the areas combined, since degeneration in the affected nuclei is hastened if they are stimulated to exhaustion. That this has been produced, is shown by a demand by the muscle of a greater current to produce contraction. The medulla as a whole may be stimulated by passing a galvanic current from one mastoid process to the other, with electrodes about four by four inches. Vertigo may ensue if the current is strong. Another method is to place the positive electrode over the base of the brain, and the negative over the motor points of the lips, the masseter muscle, and the tongue. This current is preferably a galvanic one, making from five to ten interruptions over each muscle. Stimulation of the deglutition reflex is valuable, since difficulty in swallowing is a vital loss. This may be accomplished by the faradic current with one pole on the nucha, and the other over the pomum Adami, or by the galvanic positive on the nucha, and the negative in front. A more exact method is to place a small electrode upon opposite sides of this projection, and pass a current between the two. Galvanization of the sympathetic is accomplished by placing the negative at the clavicle, close to the spine, and the positive at the nucha. This will also alleviate the stiffness of the neck. A general treatment of the whole body with faradization, or by the static breeze, has also been advised. From any form of electrical treatment we can, at the most, hope only for a retardation of the degeneration, but ionic medication is said by Little and Bokerman to have caused an actual improvement in the symptoms.

THERAPEUTICS. Always bear in mind the possibility of syphilis as a cause. The remedies advised for this condition have been based upon clinical reports of the relief of various isolated symptoms without sufficient data, in most cases, to inform us whether the symptom-complex was pseudo-bulbar palsy, which is a cortical lesion, or a bilateral one of the cranial nerve tracts, or true degenerative bulbar palsy, or myasthenia gravis. Since pseudo-bulbar paralysis is not accompanied by atrophy, it is evident that remedies marked by that symptom are inapplicable, while the contrary is true in the case of the degenerative form, which is especially distinguished by the presence of atrophy. Cure may not be possible, but there is evidence that some of the symptoms may be benefitted for a time. Remedies selected upon toxicological grounds have not proved efficient, those based upon pathological changes seem to be rather more successful, but our main indication should be symptomatology. Manganese binoxide, and Cadmium sulph. are the toxicological remedies, while Mercury is pathologically suggested. Arnica, Argentum nit., Baryta carb., Causticum, Lachesis, Nux vomica, Phosphorus, Plumbum, Stannum, and Veratrum viride should be studied for a similimum.

MYASTHENIA GRAVIS.

Asthenic Bulbar Palsy.

Definition. This is a disease characterized by great muscular weakness, affecting most strikingly the muscles supplied by the nuclei in the medulla oblongata, and which are closely related to the maintenance of the vital functions. The muscles of the limbs and trunk are also affected, and sometimes before the others. The disease finally amounts to a purely motor paralysis of the whole body, but thus far no definite causative lesion has been discovered.

The Symptoms of a Typical Case. There is a gradual reduction in strength in the whole musculature of the body of a person in early adult life. The neck first becomes too weak to hold up the head, and then the eyelids fall as by their own weight, and the eyes cannot be held parallel causing diplopia. The muscles of the face and thorax are weakened, and then of the whole body. Reflex and electrical stimulation reveal the same readiness of exhaustion. This weakness may develope to any degree, but without actual atrophy. The disease is generally fatal in from two to four years.

Differential Diagnosis. It is to be diagnosed from Progressive Bulbar Palsy, Cerebral Pseudo-bulbar Palsy, Diphtheritic Paralysis, and Hysteria.

Age. It may occur at any period from childhood to old age, from two years to seventy-two; the largest percentage of cases occurs between the ages of thirty and forty, and next between twenty and thirty. Tabulating all known cases up to a recent date, it is found that the average age of onset in men is thirty-five, and in women twenty-four.

Sex. It is generally considered that males and females are equally affected, but some statistical groups would indicate a female preponderance.

Heredity. Not a factor.

ETIOLOGY. Occupation does not seem to influence its appearance, but it has been found to follow acute diseases notably (Hoppe), and to have followed hard on continuous over-exertion (Pel). Since it is a disease of very generalized distribution, it is presumably the effect of some toxin circulating through the body, having a selective affinity for the lower motor-neuron, but as yet none has been isolated. There is some evidence, in view of the fact that a persistent thymus has been discovered in some cases that it is the result of the morbid activity of this gland. Sitzen found colloidal changes in the thyroid gland, and in nine cases evidence of an involvement of some kind or another of the lymphoid system. Pemberton concludes that it is a disease from deranged muscular metabolism involving a nitrogenous retention as notably as in true muscular wasting. Marinesco shows that the glands of the body are in a state of hyperfunction, and that the actual cause of the disease is a perturbation of the formation of antibodies to fatigue, or oxydases.

PATHOLOGY. No constant lesion of the nervous system has been discovered, but numerous observers have found lymphoid tissue in the striated (voluntary) muscles of the adrenals, kidneys, liver, and thyroid, which was not the result of inflammatory action. Marinesco agrees with these findings, adding infiltration of the hypophysis.

SYMPTOMOTOLOGY. Weakness in the muscles of the neck has been reported as the initial symptom in many cases. A case occurring in the practice of the author gives grounds for believing that ptosis and diplopia may antedate all other symptoms, and perhaps by several years. These ocular symptoms at first are transient, but finally return in a permanent form. Swallowing and chewing become very laborious, finally impossible, and continued speech readily exhausts the power of the muscles. The upper facial muscles also lose their efficiency, and the face assumes the mask-like look, which we find in paralysis agitans. After the involvement of the facial and cervical muscles, the muscles of the thorax become readily exhaustible, and those of the lower parts of the body follow in order, or are simultaneously weakened. Pure cases will show no atrophy, but it has been observed as a complicating condition. After the disease has reached its full developement, the patient will present the following picture. Ptosis has become so extreme that the upper eyelids must be held up during the day by pieces of plaster, or some other mechanical device, and fastened together by similar means, to keep them closed during the night. The initial ptosis may be of one eye, but later becomes in all cases bilateral. The ptosis may be absent on rising in the morning, but may become evident as the day goes on. At some period in all cases there will be complete ophthalmoplegia. Diplopia and strabismus are often present, and nystagmus at times. Secretion of saliva seems to be exaggerated, and the power of swallowing is very deficient, and therefore the disposition of the saliva is a constant annoyance, and often a menace to life. In spite of this condition, it is said to be a point in diagnosis between myasthenia gravis and progressive bulbar palsy that there is no dribbling of the saliva in the first-named disease, since the corners of the mouth are not relaxed and patulous. The lower jaw tends to drop, and may have to be supported, and the head rolls loosely upon the trunk, so that a jurymast is often essential to the comfort of the patient. While, as has been said, there is no atrophy, the distinctive feature is the ready exhaustibility of all the muscles of the body. In the earlier stages of the disease, muscles respond to volition, but become weaker and weaker with each contraction, until finally the part is helpless. After a period of rest they will respond again as before. In the later stages it is practically a complete motor paralysis. This ready exhaustibility is just as true when we invoke electrical excitability. It is said not to be true of galvanism, but the first response to a faradic current is prompt and energetic, the next much less so, and finally there is a complete failure, no matter how strong may be the current. The sphincters remain intact, there is no reaction of degeneration, muscle-sense and coördination are never affected, there are no pains or other sensory disturbances, the special senses are preserved, and the reaction of eyes to light and accommodation are normal. Since respiration is a constant expression of muscular energy, the effects of exhaustion here are evident at all times, and measurement of the chest will show that between inspiration and expiration there will not be a difference of more than a quarter of an inch. The involvement of the pneumogastric nucleus affects also the rhythm of the heart, and there may be changes in the secretions. Mentality is generally entirely unaffected, and they may be able to transact business with success, but some cases have been reported where there was mental excitement. Knee-jerk is exaggerated. Clonus is absent. There is speedy exhaustion of the special senses as well as of the skeletal muscles. Emotional strain and cold intensify the symptoms.

COURSE. There are remissions, and years may elapse between the initial symptoms and the final definite onset of the disease, but this is not always true, as cases have been fatal in three months. Steady progression is not the rule.

PROGNOSIS. There is a tendency to death in from two to four years. Grouping sixty reported cases, twenty-three ended fatally in an average period of eighteen months. Some cases have been reported as cured. Death occurs from inanition, choking, and inspiration pneumonia.

DIAGNOSIS. This depends upon the presence of a bulbar form of palsy, which is purely motor, and yet shows no atrophies, as a rule, nor reaction of degeneration. It is diagnostic also, that after rest the muscles are capable of strong contraction, but it is just as diagnostic that this power rapidly succumbs to the strain of repeated attempts at contraction. The point of attack is peculiar in being a ptosis, or weakness of the muscles of the neck. There are remissions of years in the early stages. Fibrillation is absent.

DIFFERENTIAL DIAGNOSIS. Progressive Bulbar Palsy has the reaction ot degeneration, fibrillation, and early atrophy. The saliva dribbles from the mouth. There is no ptosis, nor paralysis of the muscles of the neck, and there is certainly none of the muscular weakness of the whole body. It does not have definite remissions of long continuance. While neither have sensory symptoms, there are mental changes in progressive bulbar palsy, and a liability to apoplectic attacks.

Cerebral Pseudo-bulbar Palsy is the result of double, or alternate cerebral apoplexies. Mental changes are marked, and while mastication, sawllowing, and speech may be imperfect, the condition is due to spasticity, and not to weakness.

Diphtheritic Paralysis gives a similar symptomatology superficially, but in this is not a weakness, but a visible paralysis of the vocal cords, which gives rise to a loss of voice, often of respiration and locomotion, and there is a history of a preceding disease capable of producing such results.

Hysteria may simulate it closely, since while neither have fibrillation nor atrophy, they both may display muscular weakness. The weakness of hysteria is not so universal, nor has it the characteristic of ready exhaustibility to volition, and electric stimuli. Myasthenia gravis has an absence of sensory symptoms, which are marked, and almost universal, in hysteria.

TREATMENT. In general the patient must have the best of surroundings, and the most complete rest, even from conversation. Gentle massage has its advocates, and general galvanization, but not to the extent of causing muscular contractions. The most constant care must be exercised to avoid choking, as the danger is always imminent, from the inability to swallow the abundant saliva. Small swabs should always be at hand, and tracheotomy may be necessary. Feeding can be successfully carried out by the nasal-tube, if swallowing becomes ineffective, or with the patient in the intubation position.

THERAPEUTICS. Phosphorus, Causticum, Cadmium sulph., Stannum, Belladonna, Veratrum, Lachesis, Hyoscyamus, Gelsemium, and Arnica have been advised on theoretical grounds for bulbar palsies, without much regard to type. Phosphorus seems best on the whole, unless we wish to go upon a basis of stimulation. In such case Nux vomica, low, or Strychnia in any strength, and by any method seem the most promising. Stannum, high, has been reported to have helped the choking attacks, and Hydrophobinum, high, on pathological grounds, has been reported to be efficient by O'Connor. Tonics, Strychnine, and Veratrine are the physiological remedies

Calcium lactate, grs. 15, three times a day, has been advised, on the ground that it abolishes fatigue in muscles. Hoppe (White and Jelliffe) advises Citrate of Soda, grains 15, three times a day.

Hamilton reported a case (J. A. M. A., May 25th, 1912) in which thyroid feeding gave definitely bad results, but the administration of 15 gm. of thymus extract, three times a day, produced an amelioration of the symptoms, which has continued for eleven years. On the assumption of a thymus causation, X-ray treatment of the mediastinum has been suggested.

AMYOTROPHIC LATERAL SCLEROSIS.

Wasting Palsy—Spinal Progressive Muscular Atrophy—Charcot's Disease.

Definition. It is a disease of the whole motor nervous system, and while it usually is located in the cervical region, it may develope a degeneration in all motor structures, from the muscle fibres to the cells in the motor part of the cortex. The lesions on the motor side are as widely distributed as those of tabes on the sensory, but these changes are not always consecutive, as at the level of one segment the peripheral fibres may be affected, while just above it the cells may be the initial point of attack. This degeneration produces a form of progressive muscular atrophy which is peculiar in showing spasticity.

The Symptoms of a Typical Case. There is a spastic paralysis of the lower limbs, to which is added a progressive muscular atrophy in the upper part of the body.

Differential Diagnosis. Differentiation is from Bulbar Palsy, Tumor (cervical and spinal), Cervical Pachymeningitis, Syringomyelia, Myelitis (chronic), Spastic Paraplegia, Spinal Caries, Peripheral Neuritis, Disseminated Sclerosis, Chronic Poliomyelitis, Progressive Muscular Atrophy (other forms), Syphilis.

Age. It is a disease of late middle life, and the probable limits are from twenty-five to fifty. Most cases are over thirty, yet children have occasionally been subjects of it. Here it is often a secondary disease, but Gordon quotes a case occurring at the age of fifteen.

Sex. There is considerable evidence, but not a unanimous agreement, that females are most subject to it.

Heredity. This is difficult to trace, but there is in all cases a general neuropathic tendency, which manifests itself in these persons as a motor nervous system which proves itself to be too delicate to stand the strain of life.

ETIOLOGY. In all cases a faulty nervous system is universally assumed to be a necessary basic factor. There is a pronounced disagreement in the identification of any definite, exciting cause. Marie says that none is known, and Dana is in harmony with this position. Other observers of wide experience feel that simple over-exertion, or this combined with fright, exposure and deficient food, poisoning by toxic agents, such as alcohol, lead, arsenic, mercury, the acute infections, or auto-toxins are competent to excite it. Rheumatism, gout, diabetes may cause it, but the potency of syphilis as an exciting cause is generally denied. It may result from an extensive endarteritis; it is very apt to follow acute anterior poliomyelitis, and at times it has been caused by direct injuries to the back. (Gowers, Giese.)

PATHOLOGY. Collins gives the following summary of the pathological examination of a case which was under his observation for two years preceding her death. The duration of the disease was three years : (a) There was uniform disappearance of the ventral horn cells throughout the entire cord, the dorsal region being less extensively involved than the enlargements of the cord. (b) There was degeneration of a zone in the anterior fundamental columns skirting and encircling the ventral horns in the cervical and upper dorsal regions, also the anterior portions of the lateral limiting layers of the cord, expanding dorsally against the ventral and lateral border of the crossed pyramidal tract, and appearing with a comma-shaped head in the lumbar and sacral regions. (c) There was degeneration of a marginal strip of the ventral periphery. (d) There was deformity of the ventral face of the cord in the cervical and upper dorsal region, due to a concave sinking-in of the periphery. (e) There was neuroglia proliferation in the degenerative area of white matter, and also in the anterior horns of the cord enlargements. (f) There was evidence that the neuroglia proliferation was of longer duration in the cervical region than elsewhere. The degeneration was denser in this region, and had caused shrinking of the ventral face of the cord at the emergence of the roots. (g) There was atrophy of the anterior roots. (h) There was evidence of cell degeneration (chromatolysis plasmolysis, and disintegration) in the hypoglossal nucleus, and the nucleus ambiguus. (i) The pyramid tracts were intact. Starr says, "Speaking generally, a degeneration of the cells of the anterior horn is always present, and begins in the cervical region, and in sixty per cent. of the cases is confined to it, in thirty per cent. it is confined to the lumbar enlargement, and in ten per cent. involves the bulbar nuclei." The maximum degeneration of the upper neuron is in its terminals, and of the lower in its cells. In the cord the pyramidal and the antero-lateral association tracts for the motor system betray degeneration which at times may be traced clear to the motor-cells of the cortex about the fissure of Rolando (motor inhibitory of the second, and purely motor of the third layer). At other times the degeneration will die out at some one of the lower levels. As has been said, the motor nuclei of the bulb will be affected, and the nucleus of the hypoglossus is the most frequent point of attack, but the pneumogastric, facial, and motor portion of the fifth are not infrequently attacked. If atrophic symptoms predominate, we may be assured that the cells of the anterior horns have been more involved than the tracts, while if spastic symptoms prevail it is a sign of the greater involvement of the pyramidal tracts; i. e., in the former instances it is more of a lower neuron disease, and in the latter it is more upper neuron. The peripheral nerves show degenerated fibres. In any spot of degeneration an invasion of new connective tissue at once takes place, and replaces the nervous-tissue, and usually without any possibility of repair. Examination of the muscles reveals that they are shrunken and pale, have lost their striation, and the fibrillæ are separated from one another.

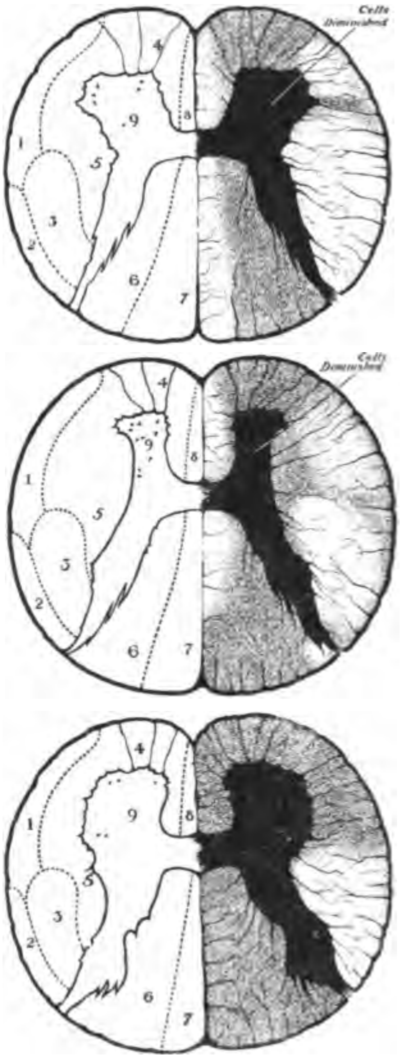


Figure 71. Amyotrophic Lateral Sclerosis.

From below upward are sections from the lumbar, dorsal, and cervical cord. In the lumbar section we see that the degeneration is confined to the Crossed Pyramidal Tract (3). In the dorsal section the Crossed Pyramidal (3), Direct Cerebellar (2), Gowers Tract (1), and Direct Pyramidal (3), and the descending group of fibres in the posterior columns. To this is added now, for the first time, a simultaneous reduction of the motor and trophic cells of the anterior horn. In the cervical section at the top we see a more complete degeneration of these tracts, and of the cells. This explains the spastic paralysis of the legs, with a wasting paralysis of the arms.

Symptomatology. Gordon (N. Y. Medical Journal, June 8th, 1907) has suggested the division of these cases into three classes. The first is the classical type of Charcot, where the main symptoms are a paretic condition of the muscles with some spasticity, then wasting of the muscles with the reaction of degeneration. In the second form there is a progressive muscular atrophy, the spastic element is not marked, but the reflexes are exaggerated. In the third form the spasticity appears first, and is very pronounced; the muscular atrophy appears later.

Keeping in mind this possible alternate character of attack, we shall best understand the condition by describing as typical an attack starting in the cervical region. The patient first discovers that he is unable to perform his accustomed physical tasks without undue fatigue. He will display a slight degree of general emaciation; not specialized as in progressive muscular atrophy. At this time he may have some rather acute pains in the arms, accompanied by tingling. Examination will reveal a general exaggeration of the deep reflexes. This is termed the first stage. After several months, in one case not for two years, atrophy begins to be apparent. This begins in the muscles of the thumb, then of the interossii, and next of the little finger. In some cases the deltoid has been the first, or after the hand, and before the arm. This atrophy proceeds very slowly, fibril by fibril, but steadily progresses, until scarcely any muscular substance is left. It is preceded, and attended by fibrillation, and when it has become complete in any part the preceding rigidity is replaced by flaccidity. The order of the atrophy is the same as in progressive muscular atrophy. The loss of power will be greater during this process than the amount of atrophy would explain, since much of the weakness depends upon a degeneration of the pyramidal tracts, which in turn, even in the absence of visible post-mortem lesions, depends upon some state of impairment of vitality in the cells of the cortex. In 1881 Beevor discovered that in these cases a jaw-jerk, and in some a jaw-clonus could be obtained, and in only one other pathological condition, viz., hemiplegia. This reflex is pathognomonic only when a mere pulling down of the jaw will produce the reflex. (Oppenheim.) This indicates that pyramidal degeneration has reached as far upward as the motor nucleus of the 5th nerve. This is called the second stage. When bulbar symptoms are developed, the disease is said to have reached the third stage, but in rare cases these symptoms have been the initial ones.

The above is not always the course, since a case has been reported where no atrophy was seen for two years, and then was only displayed in the tongue and lips, i. e., bulbar, nor is the atrophy always secondary to sclerosis in the pyramidal tracts, i. e., deuteropathic. A spasmodic drawing of the tongue, or stiffness of the cheeks or lips, may be an initial sign (bulbar). One or two cases have been reported (Schlesinger) where an Argyll-Robertson pupil has been observed, but in general the ocular muscles are not involved, although in late stages the upper facial may be so involved that the eyes cannot be closed. Some months usually elapse between the paralysis of the arms and the legs, but in six months the legs and arms have become so paralyzed that the patient is helpless, and long before that the contractures of the legs have become so great, that the patient has to push himself feebly along on the tips of his toes. These contractures are quite general, and result from the atrophy of some muscles, and the spastic over-contraction of their opponents. They are quite different from the contractures of progressive muscular atrophy. The fingers are bent in on the palm, the wrist is flexed, the forearm is pronated, while the elbow is semi-flexed, and the humerus is held tightly against the chest-wall. In other cases, owing to the atrophy of the interossii, the hand is clawed. In the lower limbs the rule is spasticity without much atrophy, so that in walking the leg is thrown around, scraping the toes upon the ground, but the flexion may be such as to force the patient to walk upon the tips of the toes. The head is drooped forward upon the sternum, owing to the atrophy of the muscles, and there is some degree of rigidity in maintaining the posture. When the bulbar muscles have become affected, we find paralysis of the tongue, lips, pharynx and larynx, and also the lower face, the upper face being spared, except in exceptional cases late in the disease. Gordon's case showed stiffness in the neck, soon followed by pain in the throat, then dysphagia, and finally dysarthria.

This wasting is preceded by fibrillation, and rigidity, and increased reflexes. In all cases, in spite of the stiffness of the limbs, the muscles are actually weak.

There are no sensory changes. Marked changes in the skin, nails, and joints do not occur.

The sphincters are not affected, except rarely, and at the very close of the disease.

Electrical reactions are mixed, as in progressive muscular atrophy. The muscles react to some degree to faradism as long as there is any muscular fibre left. To the galvanic current there is the reaction of degeneration.

The mental changes are peculiar. From the fact that the corners of the mouth hang down, the face has an appearance of deep melancholy, but actually some patients are good-natured, in spite of a realization of their fearful condition, for in one of the author's cases the mind was unclouded, and mental capacity comparatively unimpaired. They are, however, very emotional, and without reason laugh immoderately, or burst into floods of tears, and in some cases show actual feeble-mindedness.

COURSE. The duration of a typical case is from two to four years. If the attacks begin in, or quickly invades the medulla, death will ensue in two to three years. If, on the contrary, it begins in the legs, the medulla may never be invaded, and the patient may live as many as thirty years.

PROGNOSIS. Unqualifiedly bad for life over four years, unless it begins in the legs. The causes of death are practically bulbar accidents. The patient may either die from lack of nourishment, from choking from food, or by some of the mucus which is more plentifully secreted than is normal, or may succumb to an inspiration pneumonia.

DIAGNOSIS. In the typical cases there is atrophy of the upper extremities, combined with spastic paralysis of the lower; there is weakness, the deep reflexes are increased, but with the exception of some initial soreness and aching in the arms, there are no sensory disturbances, nor disturbance of the sphincters. Before atrophy there is fibrillation, and it may be made evident by exposure to cold, or percussion of the suspected muscle. In such cases the diagnosis presents no difficulties. When it begins in the lower limbs the only possible diagnosis for a time may be spastic paraplegia. Even when atrophy masks the rigidity, it may be discovered by the presence of the elbow and wrist jerk, by noting the contraction of the less atrophied muscles, and by what has been termed the "lead-pipe" character of the rigidity. In any case, even minor spastic symptoms in a case of muscular atrophy should put one on his guard for prognostic reasons.

When it begins in the bulb there may be no well-defined rigidity, or increase of reflexes; ankle-clonus may be the only indication in the limbs. There will be jaw clonus in these cases.

DIFFERENTIAL DIAGNOSIS. Bulbar palsy is held by some to be merely an upward extension of an amyotrophic lateral sclerosis, but differences may be discovered, even in cases where the bulb is invaded primarily. A true bulbar palsy does not have spinal symptoms, i. e., paraplegia, rigidities, increased knee-jerks nor ankle clonus, nor are there stiffness, cramps, or exaggerated reflexes in the muscles innervated from the bulb.

Tumor, or Pachymeningitis of the Cervical Cord. This will produce a wasting of the upper extremities, with a spasticity of the lower as the disease progresses, but its onset is marked by sensory symptoms, which are pains in the distribution of the median and ulnar nerves, and numbness down both arms, while the initial sensory disturbances of amyotrophic lateral sclerosis never are more than a painful soreness in the muscles. The onset is more rapid, and is very apt to follow an injury, and the sphincters become affected. There is objectively rigidity of the neck, instead of the falling forward, which is the more common condition found in amyotrophic lateral sclerosis. If compression of the cord by tumor lower in the cord is sufficient to produce a spastic paraplegia, it will not produce an increase in the reflexes in the upper limbs; it will give rise to sensory symptoms, and disturbance of the sphincters, as well as atrophy.

Syringomyelia. The developement is slower, and the final contractures in the hands possess a certain degree of similarity, both producing that type of deformity denominated "Claw-hand," only there are striking and essential differences. It has been stated above that amyotrophic lateral sclerosis is marked by an absence of sensory disturbance and tropho-neuroses (absolute solutions of continuity of tissue, and changes in the character of it), and by the bilateral character of the invasion of the bulb, when such extensions occur. Take up these points in succession, and observe that an absolute essential in the diagnosis of syringomyelia is the discovery of the loss of the power of perception of pain and temperament, that painless whitlow, degenerative changes in the skin and nails are usual in syringomyelia, and that when it invades the bulb its manifestations are unilateral. There is never rigidity, or increase of reflexes in the upper limbs in syringomyelia.

Chronic Myelitis may produce atrophy above, and spasticity below, if the site is sufficiently high in the spinal column, but there are bedsores and disturbed sphincters, and just at the upper margin of the atrophy there is an area of exquisite sensitiveness. Atrophy of the hand muscles is very rare.

Spastic Paraplegia cannot be diagnosed from an initial stage of amyotrophic lateral sclerosis, but it is so rare as an entity that it should usually be considered as the preliminary stage of some other denegeration of the nervous system.

Spinal Caries, if in the cervical region, produces atrophy of the hands, but there is rigidity of the spine, and hyperesthesia of it. Gibbous (a projection of the hypertrophied tissue about the bones) ought to be diagnostic; the X-ray will show the condition of the bones, soon there will be the symptoms of a compression myelitis in addition to spastic paraplegia, and the history will reveal the fact that it is a secondary disease.

Multiple Neuritis is a symmetrical disease, and always involves corresponding extremities, and usually all four of them. The onset is rapid, a few days up to a week or two, a toxic cause can generally be discovered, there are tenderness along the nerve-trunks, and marked changes in sensation, and the deep reflexes, instead of being exaggerated, are gone, or very greatly diminished.

Disseminated Sclerosis, while it has the spasticity and weakness, developes a much slighter degree of atrophy, and is sure to have some of the following distinctive cranial symptoms: optic atrophy of the white variety, staccato speech, or nystagmus, and intention tremor, which in itself is almost diagnostic of disseminated sclerosis.

Chronic Anterior Poliomyelitis, progressive muscular atrophy, and amyotrophic lateral sclerosis have been grouped together by some authors, but chronic anterior poliomyelitis develops more rapidly, a large number of muscles or a whole limb are atrophied at once, paralysis occurs first, and atrophy follows, while alterations in electrical response (a reduced reaction to currents of ordinary strength, and a peculiar slow wave of contraction in the muscle) occur before the atrophy has become pronounced.

Progressive Muscular Atrophy of other forms differs in that its course is slower, it attacks muscles one by one instead of whole segments, and it does not develope spasticities and increased deep reflexes. The myopathies occur in young children, the atrophies are preceded by hypertrophies, and the points of attack are the muscles of the shoulder or calf instead of the hand. They do not show fibrillation, nor do they invade the bulb.

Cervical Gliosis produces the same symptoms so far as it goes, but it is confined to a smaller area generally a single limb, and is much slower in onset.

Syphilis may occasionally produce similar symptoms.

Multiple Arthritis produces extreme atrophy at times, but it is not in the hands, but about joints previously diseased, just as likely in the lower as in the upper limbs; the deep reflexes are not disturbed, and the condition passes away as the joints become normal.

TREATMENT. Treatment is not absolutely hopeless, so long as the bulb is not yet affected, and a correct hygiene adds greatly to the possible benefits to be derived. Massage must be used with great care, since an over-use is an absolute detriment, and the same caution should be exercised in the application of electricity.

Warm baths combined with massage are beneficial for spasm, and electricity in the form of the constant current has been applied with some benefit to the spastic muscles to diminish the rigidity. For general benefit galvanism has been applied to the medulla, and the spine, and more specifically, where there is trouble in swallowing, apply one electrode anode stabile, over the nucha, and the other cathode labile to the region of the neck, four to five contractions. Others have thought that good results have come from thorough local faradization of the neck and spine.

Only one result may be expected from electrical treatment, and that is a relief of the spasmodic symptoms by the same treatment as is recommended for spastic paralysis. Give a descending galvanic current along the spine for about eight minutes, the strength of current averaging from twenty to twenty-five milliamperes, using flexible hand-electrodes of about three inches in diameter. After this the anode may be removed to the abdomen, and, with the cathode stabile along various segments of the spine, a current passed for two or three minutes at each segment followed by two or three interruptions. To relieve spasmodic rigidity the anode should be placed over the contracted muscles, and here a long, narrow electrode is preferable, and the current gradually raised to its maximum of about ten milliamperes, where it should be allowed to remain for two or three minutes, and as gradually reduced. The cathode is retained on the spine. Each muscle or set of muscles should be gone over in this way. Interrupted currents over the motor-points should be avoided, except very late in the disease when paresis has become a dominant feature. Doctor Steavenson, of London, recommends the electric bath.

The patient sits in the bath to which is attached the cathode, the anode being placed on the spine between the shoulders. Treatments are given of ten to fifteen milliamperes for fifteen to twenty minutes. In the majority of cases the patient feels more invigorated, spasms are very much less, and locomotion is improved.

THERAPEUTICS. It will at once be seen that in a combined sclerosis of the lateral tracts which are exclusively motor, and of the cells of the anterior horns which are motor and trophic, that only such remedies will be applicable as show a spastic form of paralysis with atrophy, and with an absence of general sensory derangement.

Argentum nit. This drug has little applicability as the sensory symptoms are predominant.

Arsenicum. (Too much sensory. Clarke.) Weak paralysis with atrophy. Skin changes indicate that it might be of use in syringomyelia. (Allen.)

Cuprum met. Attention has been directed to this drug on account of its potency in subduing spasms and cramps. It, however, has extreme sensitiveness of the spine as a prominent symptom, while the disease in question has no sensory symptoms.

Lathyrus. This is recommended. Given on toxicological grounds, and while poisonings show the production of ataxia and a spastic form of paralysis, the atrophic symptoms seem to be lacking. Clinical reports have not been favorable.

Phosphorus. Cramps in legs with weariness and stiffness have been used as indications. One case is said to have been greatly relieved by this drug in the 3x in combination with faradism.

Physostigma. This remedy and Belladonna are functional drugs, while Nux vomica is to be used on the same indications as Strychnia.

Picric acid. Muscles are readily exhausted, are spastic, and subjects of cramps. Many sensory symptoms are found in the pathogenesis which are not in the symptomatology of this disease. Use based upon a few clinical reports.

Plumbum. This seems to affect all parts of the cord equally, and so sensory symptoms are prominent in the pathogenesis, but on account of the very marked emaciation, it should be given a trial.

Strychnia may be useful for the stage of irritation, shown by spasm, which precedes the paralysis and atrophy, indicating that destruction of cells and tracts has taken place.

Sulphur. The symptoms of weakness with spasticity, and contracting spasm of extremities, are to be found in its symptomatology.

Zinc. Too many sensory symptoms.

Not much good has been claimed from physiological remedies, and cod liver oil and tonics comprise the list in any case. Some have thought that Strychnia 1-80th to 1-30th grain daily was of value, while others absolutely bar the drug on account of the aggravation of the spasticity. Patients should be taught early the use of the stomach tube, in order to combat the emaciation and anemia resulting from difficulty of swallowing sufficient food.

CHAPTER VIII.

TUMORS AND CAVITIES OF THE CORD.

SPINAL TUMORS.

Definition. The definition will include new-growths in all parts of the spinal canal, whether arising in the epidural space, on the dura, and the pial membrane, or from the structures of the cord itself, since a tumor situated in any of these structures will so soon set up symptoms pointing to an implication of all of them that anything more than a vertical localization is impossible.

Symptoms of a Typical Case. A male, from thirty to fifty years of age, gradually developes pain in a definite portion of the spine (generally in the dorsal region, or in the cauda equina), which is at first a unilateral, intercostal neuralgia, which later becomes bilateral. Next there are girdle sensations in addition, which are in turn followed by pain in certain areas of the body which are innervated from definite segments of the cord. These segments are found to correspond with portions of the spinal column which are locally painful. These pains increase through months, and sometimes a year or two, and the reflexes below this segment are increased, but are soon lost if the medullary portion of a reflex arc, i. e., cell groups in the cord, is the focus of the disease. Paraplegia next ensues, followed by a loss in sensibility below the seat of the lesion. Lastly all reflexes below the lesion are lost, there is atrophy of the lower parts of the body, which may be complete enough to produce bedsores, and more rarely there is spinal deformity, and pressure over the spine at the site of the tumor may set up a tetanoid condition.

Differential Diagnosis. This is from Caries of the Spine, Neuralgia, Carcinoma of the Vertebræ, Pachymeningitis Cervicalis Hypertrophica, Transverse Myelitis, Aneurism of the Aorta, and Sciatica.

Age. Tumors may develope at any age, but they are most common from the thirtieth to the fiftieth year; in childhood they are generally tuberculous.

Sex. Males most often.

Heredity. Multiple tumors are generally hereditary, and the fatty variety (lipoma) is usually congenital.

Etiology. Syphilis, tuberculosis, and the carcinomatous cachexia are the commonest causes, and traumatism is a common exciting cause, parasites occasionally.

PATHOLOGY. It cannot be said that any particular part of the cord is immune, but they are rather often found in the dorsal region, and in the cauda equina. There is peculiarity in the fact that when they are extra-medullary, they are rarely situated on the anterior aspect of the cord, but on the posterior or lateral surfaces. Tumors may be either extradural, intradural, or intra-medullary. Extradural tumors are infrequent, and comprise lipoma, sarcoma, cancer, teratoma, rarely myoma, gumma, and echinococcus cysts. These last are rare growths, and when found are usually situated between the dura, and the vertebræ, and are generally multiple. Lipoma is a soft, fatty, encapsulated tumor. It may originate intradurally, but usually arises from the extradural fatty tissue, and is multiple. Sarcoma, it should be said, is ubiquitous. It is often an extradural tumor, and in this case it arises from the periosteum of the vertebræ, the outer surface of the dura, or from the nerve-roots. This tumor, tubercle, and gumma, are marked exceptions to the rule that tumors of the spinal cord and brain do not penetrate the membranes, but if extradural so remain, and the converse. Sarcoma arising extradurally may not only penetrate the dura, but also the pia, and involve the cord, and the whole central nervous system following the vessels. It is often a metastatic growth, and then is often multiple. It may arise in the extradural fatty tissue, and carcinoma often does so. Teratoma is a rare growth occasionally found in this tissue. These extradural tumors vary in size from the bulk of a pea to an inch or two in length, and an inch or more in thickness. The favorite locality is the upper and lower dorsal region. They are more liable to extend longitudinally, and affect the nerve-roots, than to compress the cord. Lasting symptoms from such tumors will depend upon the rapidity of their growth. If they grow so fast that the cord is rendered ischemic, softening will result, but if the growth is slow, the cord may show remarkable powers of adjustment to the increase of pressure.

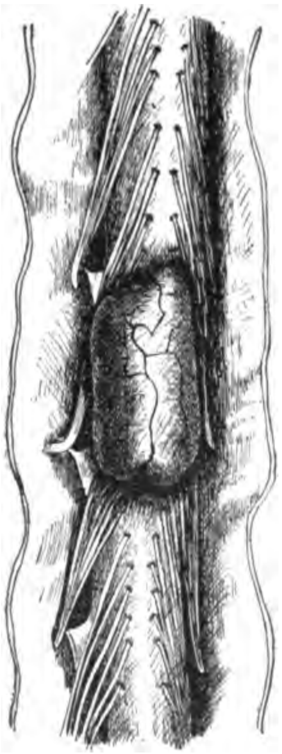


FIGURE 72. Fibroma lying on the spinal cord: removed by McCosh. (Starr.) (Courtesy of Lea & Febiger.)

Intradural tumors are the most common variety. They are less frequently the result of metastasis than the extradural variety. Fibroma and sarcoma outnumber the other varieties, but tubercle and gumma are common, endothelioma, myxoma, and psammoma are found, while neuromata may develope on the nerve-roots. Fibroma is apt to originate on the inner layer of the dura, and when it arises from that site is not liable to invade the cord itself. These tumors generally lie on the cord, and are favorable for removal. Sarcoma may develope as multiple tumors scattered over the whole extent of the meninges, from whence it often extends to, and involves the structures of the cord. Tubercle is a common growth on the meninges. If it developes on the pia it is liable to involve the cord, but the contrary is true if it arises from the inner surface of the dura. Gumma is a most generalized growth, for while it developes usually as an intradural growth, it penetrates the cord, and involves it and the meninges in a soft and rapidly growing mass. Myxoma is usually intradural, yet it may be extradural, but it never infiltrates the cord. It is single, may be encapsulated, vascular, perhaps cystic, is of slow growth, and can be easily removed. The dermoid cyst, while not exclusively a developemental fault, is usually one, and is generally found at the lower portion of the cord, and the cauda equina. Cysticerci are sometimes found here, although they are generally extradural. Psammoma is a hard oval tumor, originating in the arachnoid or dura, and lies upon the cord. It is sharply defined and chalky, grows slowly, lies on, and not in the cord, and is easily removed. These growths, except neuroma, and sarcoma, are generally single, small in size, oval or cylindrical in shape, with the long axis parallel to that of the cord, and are liable to attain their greatest size in the lumbosacral region, and about the cauda equina. They are often limited to one side, and therefore we find the resulting paralysis of the Brown-Sequard type (motor on the side of the lesion, and sensory on the other), followed by a general paralysis of the lower limbs. Extradural tumors produce symptoms in three ways; they may involve the bone, may compress nerve-roots, or may compress the cord. Intradural tumors show their effects in the last two directions.

Medullary tumors are glioma, sarcoma, tubercle, and endothelioma. Glioma is always an infiltrating growth, not sharply defined, generally distributed vertically through two or three segments of the cord, but it may extend the whole length of the cord into the medulla, and even into the brain itself. It is rapid of growth, very vascular, enlarges the cord, and is impossible of removal. Cavity formation is ascribed by some to gliomatous tumors, while others would ascribe this to gliomatosis, which is considered to be a separate condition, depending upon a developemental defect in the tissue of the central canal of the cord. In rare cases glioma may form a firm and circumscribed tumor. Glioma excepted, medullary tumors are never of great size.

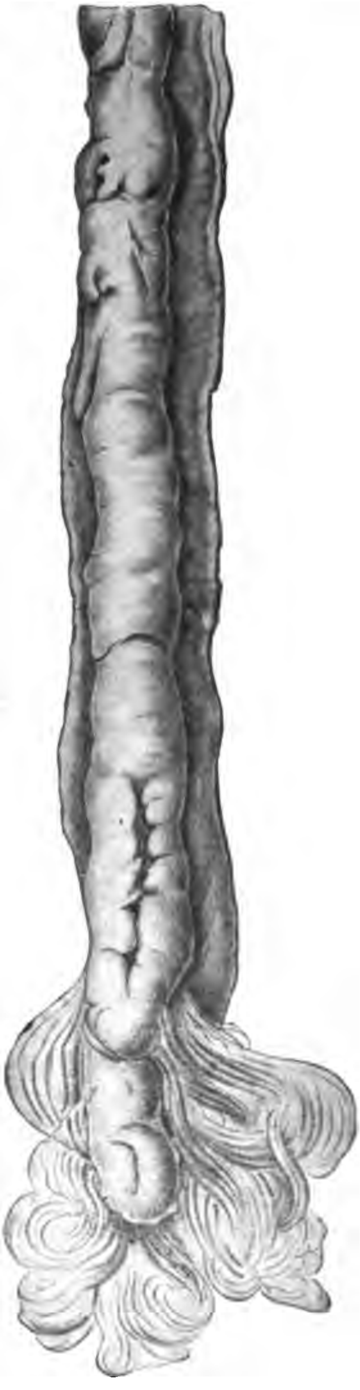


FIGURE 73. Tumor of the cord. Sarcoma surrounding the spinal cord, and invading the nerve roots. (Schulz.) (Courtesy of Lea & Febiger.)

Tubercle more often developes in the cord than invades it from the pia, and may be circumscribed or infiltrating. It is generally solitary, although it is usually complicated with tuberculous meningitis. Usually it appears as a caseous mass in the cord, although it may only erode it, and be found lying upon it, and simply compressing it. Both tubercle and sarcoma are liable to be surrounded by a small area of softening. Syphiloma are always of very rapid growth, tubercle is nearly as rapid, and perhaps more so; the same rapidity, although in less degree, is true of sarcoma and glioma, while fibroma, and myxoma, and other spinal tumors are slow in growth. The variety of tumor can rarely be recognized, the criteria being rapidity of growth, or appearance in other organs. Malignant tumors are many times as frequent as the non-malignant.

SYMPTOMATOLOGY. The disease is first manifested by the occurrence of a unilateral intercostal neuralgia (root-pain), which soon becomes bilateral. It is mild at first, but soon becomes sharp, lancinating, shooting and agonizing, and while at first is at intervals, it later becomes constant. This may be replaced by girdle-sensations. The root-pains of the early stages are present as well when the tumor is situated in the anterior part of the cord as when it is upon the posterior portion. It always arises from compression of the posterior roots. It will be entirely absent if the roots are initially destroyed, since it is their irritation preparatory to destruction which causes the pain. The segmental pains next appear, and they are due to compression of the cord. Segmental and root-pains may be combined. They are known to be signs of compression of the segments, since they are manifested in pains in the regions of the surface which are known to be innervated from definite segments. If the cord is attacked initially, anesthesia is an early symptom, and yet pain of an agonizing, lightning-like character is felt in these insensitive areas. These pains may persist for days or weeks in one spot. It is anesthesia dolorosa, like that in tabes, and the final condition after paralysis has ensued, is called paraplegia dolorosa. This is peculiarly true of paraplegia resulting from carcinomata of the vertebræ. This stage may last for months, and years even, with no further symptoms. The spinal pain is generally manifested at a point an inch or two below the site of the tumor. The nerve-trunks, with all this pain, are rarely tender.

The next symptom is an increase of the reflexes of the parts innervated from segments below the tumor with a degree of rigidity greater than is seen in any other condition. As has been said, if the tumor is situated laterally, we may find a Brown-Sequard type of paralysis at first (sensation abolished upon one side, and motion in the like area upon the other), but it is soon replaced by a spastic paraplegia. If the compression is very severe we shall get the symptoms arising from a complete severance of the cord from any cause, i. e., lost reflexes, and a flaccid paralysis. If a tumor specifically attacks an anterior root or roots before it produces any compression of the cord, we get primarily symptoms of motor irritation in the muscles to which these roots are distributed, and these symptoms are tremor, muscular spasms, contractures, and sometimes general tonic spasms with opisthotonos. Finally, in almost all cases, there is marked spastic paraplegia with contractures and deformities of the lower limbs, but unless there is the extreme compression of the cord above referred to, simulating a complete division, there is never a complete motor loss, nor a complete loss of the reflexes. When this stage of definite spastic paraplegia has been reached, the parts begin to become anesthetic, and finally completely lose their appreciation of sensation, and a band of hyperesthesia is found above that of anesthesia. There is at first an imperative desire to urinate and to evacuate the bowels, which is later replaced by an incontinence, or a paradoxical incontinence, which is a distension of the bladder with constant dribbling of the urine. If a tumor is situated in the cord itself, the parts innervated from the involved segments will be in a state of flaccid paralysis, with atrophy, and the reaction of degeneration to the electrical current. The disease may be marked by acute attacks of meningitis or myelitis, as secondary effects of the activity of the morbid growth, and in such cases the symptomatology will be modified by this fact. We may find choked disc, cranial nerve-palsies, mid-brain and cortical symptoms, viz., delirium, epileptiform convulsions, and headache due to complicating serous meningitis. There may be anesthesia of the skin with analgesia, and there may be an absence of the perception of heat and cold, or the lack of pain and temperature sensation may occur without any loss in the perception of touch. This dissociated loss of sensation is not always due to the fact that the tumor is intra-spinal, but when it occurs in extra-spinal tumors it is temporary. Herpes zoster and trophic changes are sometimes seen in the area of affected nerves. When a tumor has become fully developed, it is sometimes found that the spine over its site is tender to touch, to heat, and also to the electrical current. The characteristic pains are not absent in more than ten per cent. of the cases, and are apt to be present through the whole course of the disease. This pain in the spine is more apt to be present if the tumor is seated upon the dura than when in any other location.

DIAGNOSIS. This must be considered under three heads. First: is the cause of the symptoms a tumor? If this is answered in the affirmative the next question is this: what is its nature? Thirdly: where is it situated?

DIAGNOSIS OF THE CHARACTER OF THE DISEASE. We should be led to believe that a tumor existed, if a unilateral intercostal neuralgia developed into a bilateral attack in the same locality, if later a definite girdle sensation with agonizing constant neuralgic pain developed itself, if segmental symptoms developed in related portions of the body; and it would amount to a certainty, if at a later period a spastic paralysis developed in the parts of the body below the site of the original pain.

DIAGNOSIS OF THE VARIETY OF GROWTH. If a patient is tuberculous, cancerous, syphilitic, or has sarcoma in some other part of the body, there is a vast probability that the growth in the spine will follow the lines of the pre-existent cachexia. The rate of developement is a sign of some value. All malignant tumors are very rapid, tubercle and syphilis also, but in the two latter the history is so illuminating that other data are relatively insignificant. Benign tumors are slow, and are also peculiar in not producing any cachexia.

DIAGNOSIS OF SITE. Tuberculous tumors are generally intra-medullary, as also are gliomata. Fibromata, psammomata, lipomata are extra-medullary. Gummata, carcinomata, and sarcomata are either intra- or extra-medullary. The histological character of the tumor, and the question of whether it is intra- or extra-medullary is always a matter of some doubt. The question of its vertical level is more easily approximated. The situation of the pain is dependent upon the position of the tumor: it is segmental or root, and is felt in the related parts of the body, and not in the spine itself. The initial pain from a tumor in the cervical region is felt in the shoulder and neck, while if in the lower cervical region, is felt first in the hand and forearm. Dilatation and contraction of the pupils occur if the tumor is high in the cervical cord. The paralysis is also peculiar in being a hemiplegia at first, and later a quadriplegia (of all four extremities), the paralysis of the upper limbs being spastic, or atrophic, or a combination of the two. In a tumor of the sixth dorsal it is felt first in the nipple and chest, while if in the tenth dorsal it is first felt in the abdomen and groin. If it is the upper part of the lumbar cord, there is an early loss of the knee-jerk. A general indication of an implication of the lumbar region is a paralysis with atrophy, and all the signs of a lumbar myelitis. Severe pains in the lumbo-sacral plexus call our attention to this region. Brown-Sequard paralysis followed by a paraplegia, or analgesia, or anesthesia, or a limited muscular atrophy of early appearance indicate that the growth is within the cord. Tumors in the cauda equina are indicated by a wide diffusion of the pains, but are most marked in the pelvic region. They involve the anus and rectum, and also the sciatic nerve, and its area of distribution. It may produce a degenerative paralysis (the lower neuron type) in the area of the ischiatic plexus, but cases have been seen when the only seat of the paralysis was the bladder.

Tumors which involve the posterior tract may produce ataxia. Multiple tumor is indicated if there are several points which are the seat of intense pain with a mixed type of paralysis. This will result from the fact that one tumor may involve a reflex arc, in which case there will be an area of flaccid, degenerative paralysis, while another tumor may be located above an arc lower in the spine, and thus produce a spastic form of paralysis in the parts below it.

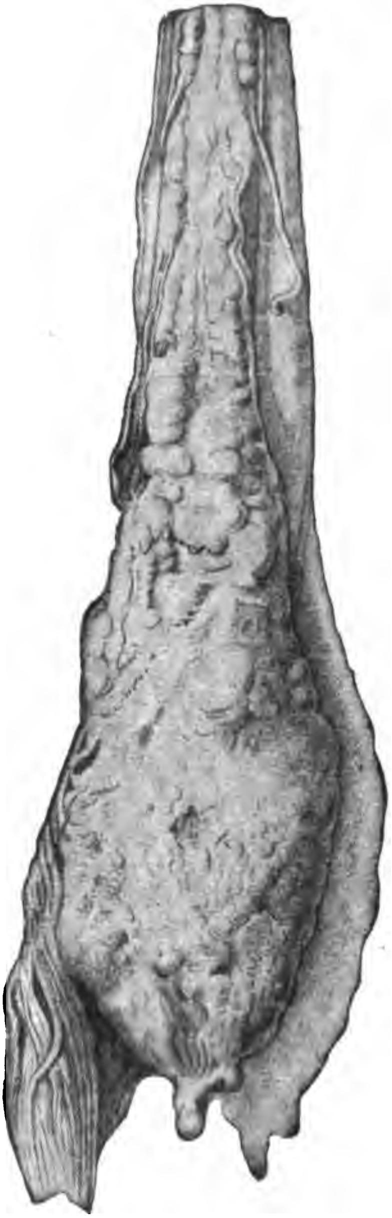


FIGURE 74. Fibrosarcoma of the lower half of the spinal cord and cauda equina. (Bruns.) (Courtesy of Lea & Febiger.)

DIFFERENTIAL DIAGNOSIS. The X-ray provides us with the most definite data for the diagnosis of disease of bone, and the presence of Caries of the Spine. Stiffness of the spine, great tenderness over the spinous processes, marked increase of pain on motion, or walking, general constitutional evidences of tuberculous disease, peptonuria, are present in caries, and not in tumor. When kyphosis appears, we know that there is caries. Pain on rotation, percussion, or jars of the spine are also signs. Symptoms are initially bilateral, and root-pains not so severe. Thickening of the vertebræ, and deformities, and pain accurately follow the nerve-trunks.

Carcinoma of the Vertebræ. This is more difficult of diagnosis, until the disease of the vertebræ is palpable, and the rapid advent of cachexia points to it. Gowers says that growths in the bones cannot be diagnosed at first, but later the pain from movements in the spine is much more intense than in tumor, unless the tumor is in the cervical region, where its detection is comparatively easy. The pain from the compression of a nerve-root as it passes through the foramen is much greater than when the compression is within the canal. The actual diagnosis must generally rest until the external enlargement betrays disease of the bone. Starr does not agree with Gowers that the pain in carcinoma is greater than in tumor, but he thinks that in carcinoma it is more commonly felt in the spine itself, as well as in the nerve distribution, and that herpes zoster is rarely seen except in tumor.

Pachymeningitis Cervicalis Hypertrophica. This produces a condition somewhat analogous to tumor, and when diagnosis is possible it arises from the fact that tumor produces initial, and unilateral disease of the nerve-root, while cervical pachymeningitis is bilateral from the beginning, and consequently pressure symptoms upon the spinal cord are more marked in tumor than in pachymeningitis. Pains in the arms are much more severe, and there is a much earlier, and more widespread impairment of all forms of sensation in pachymeningitis.

Transverse Myelitis. Usually sudden in onset, and not especially painful, unless the meninges are affected, and even in that case the pain is not in the spinal roots, but is diffuse, and is intense over the spine; bedsores, and trophic disturbances are early features of myelitis, and late ones in tumor. Severe initial pains, with paralysis as a much later symptom, suggest tumor rather than myelitis. A very chronic transverse myelitis may produce many points of similarity.

Aneurism of the Aorta. This may press upon the vertebræ, and set up necrosis, with a resulting pressure upon the cord. It becomes an intra-spinal tumor in fact. The means of differentiation are obvious but the possibility must be kept in mind.

Sciatica may suggest a tumor of the cauda equina, or vice versa. Tumor here is bilateral from the first, while any bilateral sciatic pain must be considered to arise from some growth in the pelvis, or spinal cord. The added paraplegia and root-pains in other localities, soon clear up the diagnosis.

COURSE AND PROGNOSIS. The course of the disease is usually slow, and may take some years, although the rapidity of the malignant conditions has been noted in the previous section, tubercle probably being the most rapid.

PROGNOSIS follows this order: remission has sometimes been seen, for unexplained reasons, complete helplessness with extreme pain is a natural tendency, and death is usually the result of the accompanying bedsores, myelitis, cystitis, pyelonephritis, or septicemia. Operation, while always in a sense exploratory, has been fairly successful. A cervical localization of a tumor gives the worst prognosis.

TREATMENT. This is practically surgical. When the tumor can be accurately localized, and is thought to be single, laminectomy should be performed, as the damage to the cord is quite often merely secondary to the pressure of a tumor arising from the meninges or the bones. If such a tumor is found to be inoperable, laminetomy will still conduce to the comfort of the patient by relieving the spinal pressure, and by giving the tumor equal chance to extend outward rather than inward, and reduce to that extent the injurious effects of pressure upon the spinal cord.

THERAPEUTICS. As has been pointed out, the Iodides, or a full specific treatment, have proved efficacious in some cases of syphilitic tumors, and post-mortems have shown that this form of treatment may be curative in the event of glioma. It should be employed tentatively in all varieties. There is no ground for believing that we have any specific indications for any of our remedies. It is not a condition where the symptoms are capable of very successful interpretation. If symptoms obtrude themselves which seem very characteristic of the symptomatology of a certain remedy, it should certainly be employed, and a cure is possible. The usual domain of therapeutics is entirely in the relief of pain, and this should be procured, whatever be the drugs demanded.

SYRINGOMYELIA.

DEFINITION. This is a slowly developing paralytic condition, characterized especially by a loss of the perception of pain and changes of temperature, while tactile sensation in the same area is normal. The symptoms are due to the destruction of conduction tracts and cell-nuclei, from the formation of cavities about the central canal of the spinal cord.

SYMPTOMS OF A TYPICAL CASE. A person in early adult life developes a weakness in the upper extremities, and moderate pains and paresthesias. The muscles of the hands begin to waste, and the atrophy progresses up to the trunk, until finally the muscles of the shoulder and back are implicated. Contractures take place, and we find "Claw-hand," or "Preacher's hand," the shoulders become bent, and there is lateral bending of the spine (scoliosis). The muscles of the bulbar nuclei may finally become involved. The paralysis is flaccid, and accompanied with loss of electrical reactions, impaired circulation, and trophic changes of various kinds. Later, there may be a spastic type of paralysis of the lower limbs. The order of appearance of paralysis in the lower and upper limbs depends upon the location of the cavity, or cavities, and upon the initial location, and final extension of the process. The diagnostic symptom is the appearance, with these muscular paralyses, of a loss of the perception of pain, and changes of temperature, while the perception of touch remains normal.

DIFFERENTIAL DIAGNOSIS. This is from Hemorrhage into the Cord, Anterior Poliomyelitis, Progressive Muscular Atrophy, Tabes Dorsalis, Myelitis, Tumors of the Cord, Pachymeningitis cervicalis, Bulbar Palsy, Amyotrophic Lateral Sclerosis, Spastic Spinal Paralysis, Vasomotor Neuroses (like Acroparesthesia), Leprosy (from Morvan's type), Hysteria, Dupuytren's Contracture.

FREQUENCY. Not extremely rare, but not common.

SEX. More males than females.

AGE. Twenty-five to forty. Almost always before thirty, and never after forty, except from traumatism.

HEREDITY. Doubtful, but has occurred in three members of the same family.

ETIOLOGY. It has been noticed that an injury frequently precedes the disease, as the presence of a splinter of bone, or in another case, under the observation of the author, it followed a crushing force, with resulting symptoms pointing to a hemorrhage in the cord. Syphilis has been found to produce cavities in the cord. Severe cases have developed in the absence of all exciting causes, and autopsies have disclosed beginning canalization, without being large enough to have been productive of symptoms. It is known that the glia tissue is thickest about the central canal, and also that the closure of the canal is sometimes incomplete, and that portions of the posterior septum are included, made up of embryonic cells. From all these data, the causes of this disease are considered to be due to a breaking down of embryonic tissue, the same process preceded by a growth of glia tissue, degeneration of glia tissue from vascular occlusion, or a destruction of otherwise normal tissue by a hemorrhage, or laceration. Tissues, imperfectly organized from a fault in developement, have been stimulated to abnormal proliferation by pregnancy, infections, or manual occupations involving strains to the spine, or elongation of it, which latter condition has been proven to be capable of increasing the volume of blood in the spinal cord.

PATHOLOGY. The disease usually begins in the cervico-dorsal segments, and it may extend indefinitely either upward or downward. Its most common extension is into the medulla, but it may involve almost the whole extent of the cord.

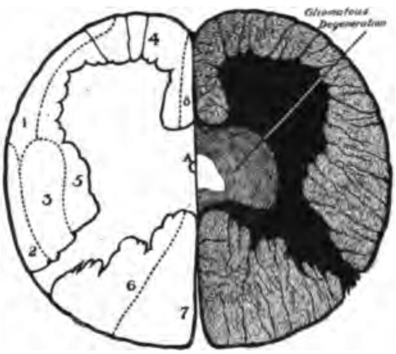


FIGURE 75. SYRINGOMYELIA.

The gliomatous degeneration, arising in the walls of the central canal, invades the gray-matter of the anterior and posterior horns to a variable extent. This gives rise, first, to a loss of the perception of pain and temperature, and, to a variable degree, to paralysis and wasting with contractures.

The post-mortem appearance is very characteristic, since the cord appears normal, or only slightly enlarged, surrounded by normal meninges, but it tears easily, and feels soft. On rupture, a clear fluid flows out, and on examination the central canal of the cord is found to be enormously enlarged; it sometimes will admit the little finger, and its outline is very irregular. The enlargement may be interrupted, and instead of the cavity being formed by an enlargement of the canal, long tube-like cavities may be found entirely independent of it the canal itself being normal. Such a cavity contains some masses of fibrin, proving it to have formerly contained blood (hematomyeloporus). At other times there will be anemic areas from compression of the spinal arteries, consequent upon a meningo-myelitis, or there will be evidences of a lymph-stasis, due to the pressure from vertebral caries. A primary enlargement of the cord, (hydromyelia) hemorrhage into the central part of the cord, (hematomyelia) or a gliomatous tumor (spinal gliosis) of the central canal, may produce similar symptoms, more or less diffused, and likely to be impossible of differentiation in a given case.

The typical condition is, however, an enlargement of the central canal, the walls of which will be the seat of some sort of gliomatous growth, which extends more or less into the surrounding gray matter. Most often it projects laterally, less often posteriorly, and least frequently into the anterior horns. It is usually not symmetrical in its extension. Occasionally it cuts off one-half the cord, giving rise to the Brown-Sequard syndrome of motor paralysis upon one side of the body, and sensory upon the opposite. The white tracts or columns are sometimes invaded, the posterior more often than the lateral, or the anterior.

SYMPTOMATOLOGY. The characteristic symptom of the disease is the fact that the perception of pain and temperature is lost, while ordinary tactile impressions are still appreciated in the same area (dissociated anesthesia). Vibration perception in the bones may be lost. Muscle and joint sensation are present, unless the postero-lateral tracts are invaded, and this is rarely the fact.

The particular kind of loss may not be complete, since the appreciation of heat may be lacking, while that of cold may be still present, or the opposite. The patient is frequently unaware of his disability, and is therefore liable to injury in his daily occupation, and if a patient displays a degree of peripheral muscular atrophy, and shows evidences by numerous scars of having suffered repeated injury with little or no discomfort, it is presumptive evidence of the presence of syringomyelia. In the early stages the loss is usually in the hands, later in the shoulders and the upper trunk, and rarely may also be in the lower limbs. The loss may be on a vertical strip on a limb, or it may be only a dissociation, i. e., heat recognized as cold, and vice versa. Sensory losses are usually found in the arms only.

TEST FOR TEMPERATURE PERCEPTION.—Fill a test-tube with cold water, not ice, since to most persons in health extreme cold feels like extreme heat. Fill a second with water of such a temperature that it can be held upon the cheek for a minute without pain; carefully dry the outer surface of both tubes. Blindfold the patient, and place not just the tip, but a considerable surface of the tube on the skin area to be tested. Leave it in position for a few seconds, as sensations are differentiated slowly, then try another area. Change from hot to cold, remembering to break the order, and do not preserve an alternation, and touch remote portions of the body from time to time, as a check.

TEST FOR PAIN. A pin is better for this purpose than a needle. Prick, alternating with touch. Change from one part of the body often enough to take the attention from the part. Make halts once in a while, and an answer when no stimulus has been given, will betray deceit, or inattention. Pain may be appreciated as a heavier pressure. The faradic brush is best of all. It is probable, from the findings in this disease, that many of the fibres conveying sensations of pain and temperature do not at once ascend in the white tracts, but run across to the opposite side by way of the anterior or white commissure, and thus gain the column of Gowers, or antero-lateral tract, in which they ascend. From this we know the lowest point at which these fibres are cut off, and can designate the particular segment injured, from charts which show what areas of the surface of the body depend for their sensory supply upon particular segments of the cord. In a late stage, tactile sensation may also be lost, but any one of the forms of sensory appreciation may exceptionally be preserved. Pain may be present.

The skin may show all sorts of vesicular, bullous or erythematous eruption, and these patients are particularly subject to numerous and extensive, but painless whitlows which are the result of repeated injuries to the tips of the fingers, which are unnoticed, on account of the lack of resulting pain. Whitlow is a lighter form, and paronychia or felon is the designation of the severer type. This epidemic of painless whitlow is characteristic of that form of syringomyelia which is called "Morvan's Disease." The skin of the extremities is usually cold and sweaty, but at times there is a total absence of sweat. The nails are diseased, and may drop off. The muscles atrophy in a manner similar to progressive muscular atrophy. It cannot be diagnosed from this condition until the sensory symptoms have appeared, since the atrophy is caused by the same lesion in both diseases; the result is the same if the sensory fibres going to the anterior horns are cut off, or if the cells in the anterior horns, lying toward the central canal, are destroyed. It is a sign that the cavity of the canal, or the gliomatous tumor which is a forerunner of cavity, has involved the trophic cells in the anterior horn. This wasting of the muscles is first seen in the finer muscles of the hand and thence slowly extends up the arm, to and involving the shoulder, and thence down to the trunk. It may begin in the muscles of the shoulder and scapula. These muscular groups are the seat of fibrillary twitchings, and show a partial reaction of degeneration. The ulnar and median groups are usually involved early, and from the unopposed action of the musculo-spiral group, there is over-extension of the wrist and hand, which tends to draw the hand into the curled-up position known as "Preacher's Hand." Spinal deformities are apt to occur, and we find scoliosis, or any other variety of spinal deviation, simple or combined, due to the fact that the asymmetrical distribution of the lesion puts an irregular tension on the spinal muscles. The face and throat muscles usually escape.

The bones are affected by an excessive fragility more often than in any other form of disease, and the joints are the seat of a painless swelling, and in this process the joint-surfaces disintegrate by a necrosis similar to that found in tabes (Charcot joints). When atrophy of the soft parts is also present, the fingers may drop off. The sphincters are not affected, unless their appropriate centers are directly attacked. Paralyses occur in connection with the atrophies in more than one-half of the cases. As the disease slowly spreads downward, we find either a spastic paralysis, with exaggerated tendon-reflexes if the pyramidal tracts are involved, or an ataxic paraplegia if the posterior columns are invaded. In this latter case the tendon-reflexes are diminished. The spastic cases are also marked by the presence of some tactile anesthesia, but of course this will not be true if the pyramidial tracts are exclusively involved, yet their involvement does not take place without the great probability of some damage to the other structures in the vicinity. If the cavity-formation extends upward, we are pretty sure to see some degree of degeneration of the cervical sympathetic resulting from the destruction of the first dorsal segment, from which it derives its fibres. In such a case the palpebral fissure upon that side is widened, and there is a reflex iridoplegia, and while the pupil is not actually insensitive to light, the eye, when it has been shaded, will react very feebly. This loss is usually one-sided. The pupils may be unequal in size. Nystagmus is sometimes present also. A laryngeal paralysis may occur, or some difficulty in swallowing, but the nerves of special-sense are usually undisturbed. It will readily be seen that there may be extensive and complete losses in the areas controlled by the nerves from the bulb and the parts above it, when we recall that structures as high as the cerebellum may be practically hollowed out by cavity-formation, and that there may be a gliomatous tumor in the pons, and owing to proliferation of the walls of the iter, there may be a resulting hydrocephalus, and resulting from that a compression of the brain-substance.

Course. The disease is a very chronic one, and may last for years. After a long term of steady progression, however, there may be complete cessation of further developement, and at any period, there may be intervals of long remission. On the other hand, at any stage of the disease, a paralysis may occur with an aggravation of all the existing conditions, such as accident depending upon the occurrence of a hemorrhage in the gliomatous new-growth, since like all other tissues composed of embryonic cells, the newly developed blood-vessels are merely canals ramifying the friable tissues.

Diagnosis. This disease is to be suspected, if we find a loss of the perception of changes of temperature and the presence of painful stimuli, but the negative element in the evidence must be insisted upon in an equal degree, viz., that in the same areas the tactile sensation must be preserved. This is necessary, since there are many conditions in which there is a loss of temperature and pain-sense, but with an equal degree of loss of all other sorts of sense-perception. If this peculiar sensory loss is found, either then, or as the disease progresses, we must also be able to demonstrate trophic disturbances of some degree in the skin, muscles, and bones, and a muscular atrophy with paralysis. The combination of any two of these conditions, the sensory loss being always one of the two, is sufficient for a diagnosis. Since the lateral and the posterior columns are often invaded, or an entire section of the cord practically disintegrated by the degeneration of the glioma, we may find cases of apparent spastic or ataxic paraplegia, or transverse myelitis, where the presence of a dissociated anesthesia denotes that we are actually dealing with an extensive syringomyelia.

DIFFERENTIAL DIAGNOSIS. Hemorrhage into the Cord can only be diagnosed from syringomyelia by its sudden appearance, and the limited area of the symptoms, since its symptoms are similar in kind.

Anterior Poliomyelitis chronica and Progressive Muscular Atrophy (especially of the Aran-Duchenne type) cannot be diagnosed from syringomyelia until the peculiar sensory changes have become so evident, that one is able to satisfy one's self that the trophic changes are due to the invasion of the trophic cells by some process originating in the central canal, rather than from a primary disease in the anterior horns, or some peripheral portion of the nutritive apparatus. Any impairment of appreciation of touch proves that the disease is not progressive muscular atrophy. Cases may exist for years without conspicuous sensory changes.

Typical Amyotrophic Lateral Sclerosis can readily be differentiated from syringomyelia from the fact that while both exhibit a paralysis, in the former the knee-jerks are never reduced, while they may be in the latter if the lesion is low down in the cord. The paralysis of the former is always spastic, while in certain localities the latter exhibits a flaccid and atrophic type. Bladder symptoms, scoliosis, paresthesias, unilateral bulbar symptoms, involvement of the trigeminal nerve, and nystagmus, are found only in syringomyelia. If the disease is atypical we must wait the appearance of the peculiar sensory symptoms.

Spastic Paraplegias. The sensory changes of syringomyelia may not always be perceptible at an early stage, but they will not be continuously absent. Such a form of paralysis is apt to arise from a syphilitic meningomyelitis, and in this we may get dissociation of sensation as well as in syringomyelia, but such a case will present different brain-symptoms. If the paralysis is of the Brown-Sequard type, the trouble is apt to be syphilitic, but if scoliosis, atrophic changes, and nystagmus (this last especially) are present, the case is syringomyelia.

Tabes. There is a great similarity at times when the lesion of syringomyelia is low in the cord. The disturbances in nutrition, even to disintegration of the joints, ataxia, loss of knee-jerk possibly, and the characteristic sensory changes may be common to both, and the diagnosis in such cases will rest upon some strong characteristics. If the upper parts of the body are relatively free from changes, except the presence of an Argyll-Robertson pupil, if there is a loss of knee-jerk and Achilles-jerk, with disturbances of the sphincters, and the sensory changes are the presence of spontaneous pains, and if a possible loss of the pain and temperature perceptions is accompanied by tactile losses in the same areas, in all probability the disease is tabes dorsalis. If on the contrary, there is increase of the knee-jerk, no disturbance of the sphincters, if there is scoliosis, if the atrophy is confined to the upper limbs accompanied by paralysis proportionate to the atrophy, and there is a loss of the perception of pain and temperature over large areas, while the tactile sense is normal in those areas, the disease is most certainly syringomyelia.

Myelitis. In either the transverse or disseminated form, this may resemble syringomyelia. Myelitis, however, usually affects the lower limbs, while syringomyelia is more often in the upper, its losses are more unequal upon the two sides, and the peculiarity of the sensory loss is generally diagnostic.

Tumor of the Cord. Tubercle, gumma, and glioma are the principal varieties. The first two are possibly diagnosed from their history. A more rapid invasion of an area limited by two or three segments of the cord, instead of five or six, and a steady progress indicate a tumor. If a tumor is larger than has been suggested, it is liable to produce the symptoms of an extensive compression myelitis, and there will be paresthesias, pain, involved sphincters, and bedsores. The Brown-Sequard syndrome is more probable in tumor than in syringomyelia.

Pachymeningitis Cervicalis Hypertrophica. This disease runs a more rapid course, there is local tenderness in the spine, the head is kept in a fixed position, there are root-pains of a very pronounced character, extending to the upper chest, the shoulder and the arms, there is less atrophy, but a more pronounced reaction of degeneration. The anesthesia also is for all forms of sensation, and it is distributed in smaller areas.

Bulbar Palsy. Syringomyelia may develope bulbar symptoms, but it is long after the arms have shown their peculiar atrophy and paralysis, and the legs have become somewhat spastic. Bulbar palsy begins in the bulb, and all the symptoms common to the two diseases are later additions.

Caries of the Spine. At a late stage of this disease, we may find symptoms which remind us of a syringomyelia, but it has been the outcome of a compression or secondary myelitis. The sensory disorders are rarely of the proper type, and the history of tuberculosis, or some of its indications, can be discovered, and the X-ray discloses changes in the vertebræ.

Leprosy. This resembles that type of syringomyelia called "Morvan's Disease," but neither in the cutaneous lesions, nor in the cord post-mortem, can the bacillus be discovered in syringomyelia. Another point of difference: in "Morvan's Disease" the plaques are upon the upper extremities alone, while in leprosy they are also upon the face and legs.

Hysteria. This may show the sensory losses peculiar to syringomyelia, as it does those of other diseases but the stigmata of hysteria are apt to be present. Failing these, the patient is apt to be younger, of the female sex, the disease comes on over night, or in a very few days, and some emotional strain, either sudden or prolonged, is the evident cause.

Dupuytren's Contracture seems probably to be actually an incomplete syringomyelia.

PROGNOSIS. The disease is a progressive one, and cure is very improbable, but it may be a long time before it is disabling. Some cases seem to have been arrested, and long remissions are not infrequent.

TREATMENT. The disease does not seem amenable to treatment, when we refer to a restoration to former health, but there are many conditions which must be cared for, and attempts must be made to aid nature in the halts which sometimes occur, or in the total cessations, which are a rarer contingency. Hygiene and massage will conserve the muscles, and electricity will aid in this, and will also benefit the atrophies, whether they be general or local. The possibility of favorably influencing the resolution of the whitlows by the negative galvanic electrode (stabile) should also be kept in mind. Beaujard and Lohermitte report favorable results by the use of the X-ray, but Hare says it lacks confirmation. The treatment is by irradiation through the laminæ of the vertebræ, alternating the two sides. The areas of analgesia should be protected from harm, and the slightest injuries should be treated with the most rigid aseptic precautions. Use apparatus for the scoliosis.

THERAPEUTICS. There is little to guide us here, but for all atrophic conditions Phosphorus 6th would seem best indicated. Some have advised Plumbum met., Cuprum met., Zinc. met. (upon the results of poisoning by the metal), Arsenicum, Fluoric acid, Argentum nit., Physostigma venenosa, Belladonna, Nux vomica, and the anti-psorics. For the whitlows O'Connor speaks of Dioscorea, Natrum sulph., Silicea, Sulphur, Hepar sulph., and Calcarea, but the whitlow of syringomyelia is painless, while the symptomatology of all these remedies is so very rich in sensory symptoms, that their use must be entirely experimental.

CRANIO-CEREBRAL TOPOGRAPHY.

Successful surgery of the brain has been based upon an ability to localize the fissures and convolutions of the brain by utilization of landmarks upon the external surface of the cranium. Since the advent of asepsis, it has been possible to raise safely large bone-flaps, and identification can be made by direct inspection instead of by rather delicate external measurements, which may be somewhat inaccurate, if the cranium in question is unusual in some of its proportions. About all that is necessary at the present time is to recognize the level of the base of the brain, the posterior limits of the frontal lobes, the exact position of the fissures of Rolando and Sylvius, less often the parieto-occipital fissure and the position of the lateral ventricles. Verifications of a motor area may be made by touching the supposed area with a metallic faradic electrode before the dura is incised.

Rolandic Fissure. Pass a tape over the vertex between the nasion and the inion. At a point 55.7 of the distance back from the nasion, make a mark through the scalp into the cranium. From this point drop a line downward and forward at an angle of 67½° with the line from the nasion to the inion, and it will lie over the fissure, which is 3.3 inches long in an adult, and 2-2½ inches long in a child. Another method of finding the lower end of the fissure is to erect a line vertically from the upper border of the external

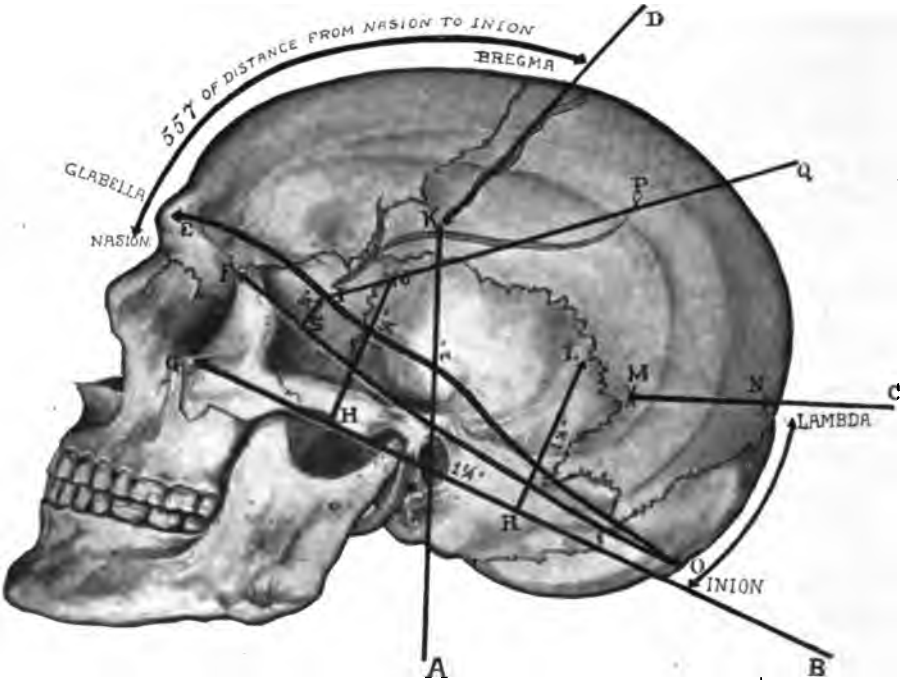


Figure 76. Cranio-Cerebral Topography.

K, D, Rolandic Fissure; J, I, P, Q, Sylvian Fissure; M. N, C, Parieto-occipital Fissure; A, K, perpendicular through External Auditory Canal to mark foot of Rolandic Fissure; G, H, R, B, base line upon which are erected perpendiculars H, I, and R, L for locating ventricles; F, O, base line upon which is erected perpendicular S, I, for localizing Anterior end of Sylvian Fissure; E, O, Lower margin of Brain.

auditory meatus. A point two inches above the meatus measured upon this line marks the lower end of the fissure of Rolando.

Fissure of Sylvius. Draw a line from the external angular process of the frontal bone to the occipital protuberance by the shortest route between these two points. The line will droop a little toward the external auditory meatus in avoiding the great convexity of the skull, which convexity lies in the course of a direct horizontal line between the two points. Such a curving line lies about a half inch above the meatus. A point on this line 1½ inches posterior to the external angular process marks the commencement of the fissure. A straight line from this point to the center of the parietal eminence accurately outlines the posterior limb of the fissure, and in part closely conforms to the squamo-parietal fissure. The ascending limb of the Sylvian fissure corresponds closely with the squamo-sphenoidal fissure in its entire length, and is continued upward in the same line for one-half inch. To expose Broca's convolution trephine immediately in front of the great wing of the sphenoid. (Keen.)

Midlle Fossa. The floor of the middle fossa corresponds very closely to the line indicating the posterior limb of the Sylvian fissure at the point where it passes the external auditory meatus.

Frontal Lobes are practically covered by the frontal bone.

Parieto-occipital Fissure. This is found by first finding the lambda, which if not evident by palpation, can be located by measurement, as it is a point 22.8 of the distance from the inion to the nasion on the nasio-occipital line, mark a point 3 m.m. in front of it; draw a line at right angles to the saggital through this point, and then measure about 2.5 cm. on this line, either side of the saggital point.

Ventricles. Draw a base-line through the lower border of the orbit and the external auditory meatus. At a point on this line prolonged 1¼ inches behind the meatus, erect a vertical line, and make a mark on it 1¼ inches above the base-line. Trephine at this point, and plunge a trochar toward a point on the opposite side of the cranium 2½-3 inches above the external auditory meatus of that side. The posterior horn of the ventricle will thus be reached at a depth of 2-2½ inches. Using the same measurements in front of the external auditory meatus, and directing the trochar toward the same point of the opposite side of the cranium, we can reach the anterior horn of the ventricle.

CHAPTER IX.

DISEASES OF THE BRAIN AND ITS MEMBRANES.

MALFORMATIONS OF THE BRAIN.

The brain is subject to a number of malformations, some of these affecting the brain alone, and that perhaps in part only, while others are gross teleological faults, affecting the cranium and its contents. Anencephaly is a condition where the cranium has failed to develope, and the partially formed cavity, owing to a simultaneous lack of developement of the brain, contains only a dark and vascular mass of membrane. Such children cannot live. If there is only a greater or less defect in the cranium, due to a failure of the bones to unite, or to sufficiently develope, a condition arises which is called Encephalocele, if the membranes protrude carrying along the brain-substance, while if the sac is of membrane only the condition is called Meningocele. There is usually a mixture of the two conditions. The dura is wanting in this envelope, and the covering is developed from the pia-arachnoid, and the subarachnoid space is filled with fluid. Under these conditions the brain defects are such that the patient is a hemiplegic, with contractures as a general result, and is a deaf mute with some grade of imbecility.

In the intra-uterine life of the child an accident to the mother, or her diseased condition, acute or chronic, may retard, or entirely stop the developement of some part of the brain of the fetus. On the other hand, it may destroy parts of it which have already gone far toward perfection of structure. All these deformities depend upon some pathological condition of the circulatory system, and the variation in results depends upon the site of the lesion, the gravity of the changes, and very greatly upon the degree of developement which the fetus has attained. The slighter degrees of injury are the cause of the trifling asymmetries of structure which are so common, and which have a very slight effect upon the appearance, or the comfort, or the efficiency of the person; a severer grade produces the so-called Birth, or Congenital Palsies, which are described elsewhere in this book; the severest type produces the microcephalic idiots, short-lived as a rule, or the monsters who die in a few hours or days.

Microencephaly is diminution in the size of the brain, and with some degree of replacement of the nervous tissue by an overgrowth of the neuroglia The cause is most probably an encephalitis which induces a starvation of the brain.

There has been some credence given to the theory that it is the result of a premature ossification of the sutures of the skull, the brain thus constricted being debarred from further development. In this condition a lobe, some of the basal ganglia, or the corpus callosum, may be found to be absent.

Microcephaly is the condition where the skull and the brain are both pathologically undersized. The brain of a normal adult male weighs about 1375 grains, that of a female 1245 grains, while the microcephalic brain will weigh from 900 down to 200 grains. An adult cranium which measures less than 43 cm, in its great circumference, contains a microcephalic brain (Dana).

Microgyria. Sometimes the normal fissuring of the cortex of the brain is replaced by delicate plications resembling the surface of the cerebellum. The result is a great multiplication of the number of convolutions with a corresponding reduction in their size. While it is sometimes a local defect it is generally universal, and is often found in cases of microcephaly.

Cyclopia or Synophthalmia is a fusion of the two eyes in a single mass which occupies an undivided cavity, resulting from a failure of the anterior cerebral vesicle to divide.

Macrocephaly is an hypertrophy of the brain in all its parts, and the skull is also enlarged. It is supposed to be the result of alcohol or syphilis in the parents (Gordon), and imbecility, idiocy, and convulsions have been observed in such individuals.

The cerebellum is subject to similar malformations to those described as affecting the brain.

CEREBRAL PACHYMENINGITIS.

DEFINITION. This is an inflammation of the dura of the brain, and is a much more rare affection than an inflammation of the pia. It most commonly affects the thick external layer alone, and it is then termed pachymeningitis externa, and less often the thin internal layer, when it is called pachymeningitis interna. It may be acute, or chronic, purulent, hemorrhagic, or hypertrophic, and sometimes is serous. The two primary divisions will be separately described.

CEREBRAL PACHYMENINGITIS EXTERNA.

DEFINITION. It is an inflammation of the external or periosteal layer of the dura, which is almost universally secondary to injury, or disease of contiguous structures. It may be hypertrophic, or purulent.

SYMPTOMS OF A TYPICAL CASE. A person who has been suffering from an injury of the cranium, or an inflammatory disease in scalp, face, ear, nose, or orbit, becomes comatose, or has convulsions of the Jacksonian type, followed by paralysis of the monoplegic variety. This paralysis is remittent, and unaccompanied by sensory disturbances, or aphasia. The last stage may be marked by one-sided optic neuritis.

DIFFERENTIAL DIAGNOSIS. This is from Tumor, Syphilitic Meningitis, Tubercular Meningitis, Cerebral Apoplexy, Cerebral Thrombosis.

AGE. Very rare in childhood.

ETIOLOGY. A severe injury to the cranium is extremely likely to involve the external layer of the dura; obviously certain to result from fracture, or penetrating wounds of the skull. If caries, or necrosis of the bones of the skull results from disease of the middle ear, the nasal cavity, or the orbit, the dura is usually implicated. A syphilitic new growth of the skull is a common cause, and erysipelas of the face and head may excite it by way of the veins of the diploë.

PATHOLOGY. If the exciting cause is a non-infected injury, it will result in the hyperplastic form. There will be an abundant cell-proliferation, and the membrane will become thickened, and adhere strongly to the bone, instead of being easily separable as in health. This condition generally is true of a small area only. If the exciting cause is an injury with infection, or an extension of a pre-existing infection, we find the purulent type, limited to a small area, as is true of the preceding type. In this case we find the dura separated from the cranium by a collection of pus, practically an abscess of the dura. This process may infect the whole structure of the dura, and involve the pia in a general infection, which will result in a matting together of all the membranes. In other instances the collection may become so great as to produce a bulging of the dura.

SYMPTOMATOLOGY. In general it will not be easy to separate the symptoms arising from the original disease and those additional ones due to the subsequent implication of the membrane. Usually, however, we shall find a more pronounced and constant headache, and if there is a collection of pus, the pushing inward of the membrane will practically amount to a tumor, and will exert pressure upon the brain, and may produce weakness or paralysis on the opposite side of the body. Such a projection will also interfere with the circulation in the vessels lying below it, and will disturb the functions of the brain in the areas to which these vessels are distributed. If the meningitis occurs at the site of a sinus, it will induce a primary sinus thrombosis by an extension of the inflammatory process, or by simple compression. A rise of temperature dependent upon the extension to the meninges is rare.

COURSE AND PROGNOSIS. The condition is essentially chronic, although it may be acute in the purulent type. The prognosis is serious, but less so than in the other forms of meningitis.

Diagnosis. Rarely diagnosed during life. It is to be inferred from an increase in the gravity of the symptoms of the primary disease. It has a large basis of probability if to the symptoms of the initial disease are added mental disturbance, and is quite certain when the opposite half of the body gradually developes some grade of paralysis.

Treatment. The treatment is distinctly surgical, and may be addressed to the primary process, or if the area is accessible, it might extend to the drainage of the purulent collection, or to the clearing out of the thrombosed sinus.

Pachymeningitis Interna.

(Hemorrhagica, or Hematoma of the Dura.)

Definition. This is a chronic inflammation of the inner layer of the dura, either serous, purulent, or hemorrhagic.

Symptoms of a Typical Case. No typical symptoms can be given.

Differential Diagnosis. This is from Tumor, Syphilitic Meningitis, Tubercular Meningitis, Cerebral Apoplexy, Cerebral Thrombosis, and Abscess.

Age. It is found in very early life, and also in late life.

Sex. It is more frequent among males than females.

Etiology. The serous variety is very rare, but is found in young children, in dementia paralytica, and in chronic hereditary chorea; i. e., in diseases diminishing the bulk of the brain, and is sometimes the result of the congestion of the meninges by sun-stroke. It is called external hydrocephalus. The purulent variety is also rare, and may be the primary result of infection, but is more likely to be the result of an extension from the external layer of the dura, or from the pia. The hemorrhagic variety vastly outnumbers the others, and is the result of an accidental rupture of a meningeal artery which has been weakened by the changes incident to old age, phthisis, cardiac, and renal disease, the exanthemata, erysipelas, and cachectic and purpuric states. It principally occurs in the course of chronic alcoholism and the dementias, especially in the paretic type, but both Boeckman and Wohwill, from the analysis of ninety-four cases, conclude that the potency of alcoholism in the production of this condition has been very much exaggerated. They consider that the hemorrhagic form is due to some preceding lesion of the endothelium.

Pathology. The serous form results from an exudation from the vessels, and probably minute capillary hemorrhages, which result in an extremely thin membrane over the inner surface of the dura, and an increase in the free fluid in this locality, which is normally only sufficient to moisten the surface. The purulent form shows adhesions of the membranes, and greater or less accumulations of pus. The hemorrhagic variety shows a condition which is the result of hemorrhage and hyperplasia. It is not at yet decided whether a toxin has first caused a proliferation of the epithelial layer of the dura into a tremendously increased bulk which is traversed by frequent blood-channels which break down, and leave hemorrhagic spots, or whether the vessels first break down, and the layers of fibrin are the result. However initiated, the result is a fibrinous deposit, layer on layer, sometimes as many as twenty. which being full of blood-channels with poorly organized walls, is the seat of repeated hemorrhages. The new hemorrhages, as they are called, separate the older layers, or form new ones on the surface toward the brain. A mild case will produce a thin, pinkish deposit on the dura which may easily be peeled off, but typically there is a tenacious mass, with perhaps twenty layers, marked with the yellow and brown discoloration of recent and old hemorrhages. There may be a mass as large as a goose egg. As a natural result, the bones of the skull may be thinned out by pressure of the new growth, or on the other hand, the cerebral convolutions may be flattened. In children this increase of cranial pressure will retard the closure of the fontanelles, and the ossification of the sutures. This condition is usually found in a circumscribed patch at the vertex, in the distribution of the middle meningeal over the motor zone, but occasionally in the basil fossæ. It may extend over one or both hemispheres.

SYMPTOMATOLOGY. Symptoms may be absent, and are generally indefinite. This pathological condition, however, has been found post-mortem in cases presenting now some, now others, of the following symptoms. A prodromal period of excitement with physical restlessness. This is followed by a severe headache, which is in turn followed by a coma, which may persist with a retarded pulse for days and weeks. This coma may be intermittent, a peculiarity which marks many of the symptoms. Instead of coma there may be apoplectiform attacks, focal or Jacksonian epilepsy, or localized spasm, or, less commonly, convulsions. The headache may be limited to the vertex, or temporal region at the onset, and it may be accompanied by vomiting. In the coma, paralytic symptoms are apt to develope, and maybe monoparesis, or of one side afterwards spreading to the other. This paresis may be accompanied by rigidities and contractures, or may be free from them. The paralysis is remittent. There are awkwardness in movement, loss of memory, and of intellectual capacity. Sensation is rarely affected, nor is aphasia often present. Conjugate deviation of the eyes has been observed, and also nystagmus, while optic neuritis, and choked disc, often of the opposite side, are liable to appear in the final stages.

COURSE. It is usually an incident in a chronic disease, and since the early symptoms are not diagnostic, we have no data from which to detect the date of its origin. For a long time there will be indefinite symptoms of illness, but later there will be apoplectiform seizures, or comatose conditions for a limited time, which will always leave certain vestiges behind in the shape of weakened intellect, or some degree of paralysis.

PROGNOSIS. Rarely cases have terminated in recovery, with an absorption of the exudate; but usually there is an incurable primary condition, of which this is a secondary result, and, therefore, the ordinary conclusion is death.

DIAGNOSIS. Rarely diagnosed during life. If, however, a case of chronic alcoholism, or general paresis developes a chronic weakness of the limbs, and mental failure with more or less of the symptomatology detailed above, we should be justified in hazarding a diagnosis.

DIFFERENTIAL DIAGNOSIS. Tumor. This subdural thickening practically amounts to a tumor, and no distinction can be made, unless the etiological basis for pachymeningitis interna is very evident.

Meningitis of any type is difficult to distinguish while the patient is comatose.

Syphilitic Meningitis is to be diagnosed from the prominence of the nocturnal headache, and symptoms of specific disease in other parts of the body, and a history of exposure, or habits which would suggest its great probability.

Tubercular Meningitis in Children. This differs from pachymeningitis interna in the presence of constipation, retraction of the abdomen, severe headache, rigidity of the neck, and marked troubles with respiration and circulation. The cranial nerves at the base, except the optic, are spared in pachymeningitis.

Cerebral Apoplexy. The possibility of confusion would exist only in the case of adults, and even then, the rapidity of the onset, and the greater permanence of the results would give us grounds for differentiation. In pachymeningitis there are intercurrent convulsions, and frequently choked disc.

Cerebral Thrombosis. This may easily be confused, since the symptoms in their gradual onset, and transitory nature, are quite similar.

Abscess. The symptoms which may possibly differentiate abscess from meningitis is the fact that the pain of meningitis is superficial and localized, and is not aggravated by motion, which symptoms are the opposite of abscess. The fever is also a less constant feature of abscess.

TREATMENT. This is identical with that for the inflammation of the same membranes in the spinal canal, and, therefore, will be found at the end of that article.

ACUTE CEREBRAL LEPTOMENINGITIS.

DEFINITION. This is an acute inflammation of the Pia-arachnoid covering the brain, and in about one-third of the cases it extends into the meninges of the cord. The meninges are not always affected as a whole, but in such definite areas that we are forced to recognize a more or less defined localization upon the Convexity, at the Base, and in the Ventricles, as well as a diffuse form which is called Cerebro-spinal. There is also a diversity in the pathological results of the various infections, and so the cases are again divided into the Serous, Purulent, and the Tuberculous, and again according to the particular infecting agent, Meningococcic, Pneumonic, etc., and from the Tubercle Bacillus, and Syphilis.

THE SYMPTOMS OF A TYPICAL CASE. Except in tuberculous cases there is an initial fever, with an initial delirium, with the speedy addition of a severe headache, which lasts through all stages of the disease. It is marked by vomiting, and a rapid prostration of the mental and bodily powers. There is constipation, but urination is involuntary. Inflammation of the optic disc (papillitis) often ensues, and the other cranial nerves are more or less affected, so that sight and hearing are usually impaired, and strabismus is an almost constant symptom. The irritation of the cortex is shown by the presence of Kernig's Sign, and the vasomotor paralysis by the "tache cerebrale." Irritation of the upper cervical nerves produces retraction of the head, and when the lower spinal nerves are irritated there is curvature of the spine.

DIFFERENTIAL DIAGNOSIS, from Typhoid Fever, Febriculæ of Children, Rheumatism, Muscular (nuchal) Rigidity from other causes, Intra-cranial Tumor, Hydrocephaloid of Marshall Hall, Hysteria, Meningeal Hemorrhage, Meningism.

AGE. While it may occur at any age, the greatest number of infantile cases occur between the third and sixth month (Barlow's statistics from Starr), and the majority of all cases occur during the first and second years of life. Children, more often than adults, suffer from the Cerebro-spinal form; Septic forms occur at any age, and Tuberculous from two to fifteen years most frequently.

RACE AND HEREDITY are not factors.

ETIOLOGY. Osler (Cavendish Lectures, 1889) classifies the causes as follows. They are first divided into Primary and Secondary:

PRIMARY.

(1) Cerebro-spinal Fever.

Which is either Sporadic or Epidemic. Caused by the Diplococcus intra-cellularis.

(2) Pneumococcic.
  Meninges alone involved, or share in a general pneumococcus infection.
  Caused by Pneumococcus.

SECONDARY.

(1) Tuberculous.
  Caused by Bacillus Tuberculosis.
(2) Pneumococcic.
  (a) Secondary to Pneumonia, Endocarditis, etc.
  (b) Secondary to injury of the cranium or its fossæ, or to disease. All caused by the Pneumococcus.
(3) Pyogenic.
  (a) Following local disease of the Cranium, or a local infection elsewhere.
  (b) Terminal infection in various chronic maladies. Both *a* and *b* are caused by various forms of Staphylococci and Streptococci.
(4) Miscellaneous acute infection.

In Typhoid Fever, Influenza, Diphtheria, Syphilis, Gonorrhea, Anthrax, Actinomycosis, and other acute diseases, caused by the Typhoid bacillus, Influenza bacillus, Diphtheria bacillus, Spirocheta pallida, Gonococcus, etc.

PATHOLOGY. Infection of any form can reach the meninges, either from injuries which penetrate the cranial vault, and lay them open to external causes of infection, or from near-by foci of purulent affections of the ears, nose, frontal sinuses, etc.

The most common source of such infection is purulent disease of the middle ear, or of the cells of the mastoid, which conditions are common results of influenza. The other source of infection is from micro-organisms in the general circulation. The primary effect of such infection is to thicken the coats of the vessels, and infiltrate them by proliferation of round cells derived from the fixed connective tissue, and by a migration of leucocytes. The vessels may finally rupture, but in any event the meninges are thickened by this exudate, the lymphatics are plugged with débris, and, moreover, the exudation from the surface in certain areas binds the arachnoid and pia and dura together, which should be free to move upon one another without resistance, since their function is simply to support without impediment the blood and lymph channels supplying the brain. In purulent cases the exudate becomes a yellowish green and sticky fluid, which is most abundant along the sulci, but more or less over the whole surface of the meninges. It insinuates itself along the sheaths of the cranial, and upper spinal nerves, and thus inflames them. Owing to the rupture of arteries, and plugging up or obliteration of veins and lymphatics, the superficial layers of the cortex are the site of serous infiltrations, small hemorrhages and foci of pus. While a purulent meningitis confined to the ventricles is said to occur sometimes in childhood, such an invasion is to some degree a feature of all cases. It is due to a congestion of the choroid plexus, or an inflammation of the ependyma, from which results an exudate similar to that upon the cortex, by which the foramina of the ventricles are occluded, and a condition of hydrocephalus results, but less often than has been supposed. The cranial nerves are not all equally affected. The third nerve is especially liable to invasion, and, therefore, strabismus is a most constant symptom. The paralysis of the facial and optic are next in frequency, while the olfactory almost always escapes. This purulent exudation is lacking in the meningitis of infants, and also in the tuberculous form, but is replaced by a serous exudate composed of serum and lymph, which thus appear as flocculi in the thickened cerebro-spinal fluid from lumbar puncture.

SYMPTOMATOLOGY. The symptoms of the various varieties of inflammation of the pia-arachnoid have so many general symptoms in common, that it seems best to describe the condition as a whole, modifying it from time to time in points where specially marked variations occur. It will, however, not be amiss to review briefly a few facts showing how the localization is affected by the etiology, and flowing from that, how the symptomatology is, to a certain extent, dependent upon broad differences in localization. For instance: the epidemic form affects the entire pia, both cerebral and spinal, while if it is a secondary disease, dependent either upon injury, or foci of infection within its immediate vicinity, we shall find a septic form which has a peculiar predilection for the meninges of the convexity, and perhaps only a small portion of this, even; the base may entirely escape.

There is what has been called Posterior Basic Meningitis of Infants. M. Wollstein, of the Rockefeller Institute, found no difference between its organisms and those of the Epidemic Cerebro-spinal form.

The tuberculous form attacks the base as a whole, from whence arises the frequent synonym for this form, namely, "basilar meningitis." If syphilis is the primary cause of the meningitis, we shall most certainly find the seat of its sole or greatest activity at the base, near the posterior perforated space, and spreading thence over the crura cerebri, and the sides of the medulla and pons.

What variation of symptoms should we reasonably expect from these variations in localization? If the convexity is affected, delirium, local convulsions, and hemiplegic weakness are common, vomiting is somewhat rare, palsies of the cranial nerves and optic neuritis, for the most part, are lacking. Localization within a limited area of the convexity would be indicated by monoplegia. If the meninges at the base of the brain are the seat of inflammation, the cranial nerves suffer early, vomiting is frequent, optic neuritis usually developes before the symptoms have reached a high degree of intensity (late in other forms), and delirium comes later. We may draw the conclusion that not the whole, but only a part of the basal meninges are implicated, when we find the cranial nerves affected upon one side only. When retraction of the head is an early and strongly marked symptom, it implies that the membranes about the pons and the medulla are affected, although the symptom is actually caused by irritation of the cervical nerves. No one of the above localizations excludes the possibility of ventricular damage, since if the disease is of the convexity, the inflammation may extend into the ventricles by following the infolding of the pia (velum interpositum), while if of the base, the foramina of communication between the ventricular and the cerebro-spinal space furnish an avenue by which infection may enter the ventricular cavities, and in addition produce indirectly a hydrocephalus by an adhesive inflammation about these openings. A primary meningitis of the ventricles is said to be possible, but its symptoms are not distinctive, except that the cranial nerves are not generally affected. All cases of cerebral leptomeningitis, whatever be the cause or localization, will present, to some degree, the following conditions and symptoms.

Headache. It is generally an initial symptom, although some purulent cases, and those, to some degree, of a serous character from altered states of the blood may not exhibit it. It is generally frontal, or occipital, but may be diffuse. It is peculiarly unmanageable, and lasts throughout the disease with such intensity, that while the patient is conscious he complains of it bitterly, and even in coma the face may be seen to scowl with the suffering. Its intensity gives rise to the shrieks which are known under the name of the "hydro-cephalic cry," and causes the patient to rub the head upon the pillows, or continually grasp it with the hands.

Vertigo. This occurs at times, and persists as long as the patient is conscious, but it is often obscured by other symptoms, so that it is not noticeable.

Delirium. This comes on in a short time, varying from a few hours to as many days, according to the severity of the process. The patient shows from the first the effect of the disease upon the sensorium by being deficient in the power of attention, and his replies are short and irrelevant. It is very variable in degree, and is usually low in grade, but from the mixture of pain and intoxication, it may be wild, or busy, like that of typhoid or alcoholism. As a diagnostic point, its value rests upon its association with headache. At first it may be only at night, or in sleep, but once developing, it is persistent.

Vomiting. Cerebral in type, i. e., projectile, without previous nausea, and at first with a clean tongue, which later becomes foul, and with a bad breath. The stomach is simply intolerant of food. It may be a very early symptom.

Temperature. The type of disease is no more diverse than the degree of the rise in the temperature, and this will be specifically stated later. In the typical form, however, while a rare case developes no fever, it will steadily rise until it attains a height of 104° in a few days, and toward the end of a fatal case it will go to 108,° or, on the contrary, become subnormal.

The Tuberculous form may show a low fever and that only toward night in its incubative period, which may extend over some weeks. Even in cases where the temperature is high, it does not always show an evening rise, nor is the final elevation regular in its developement, but fickleness is its characteristic. These irregular variations are ascribed to the developement of some nervous conditions, which neutralize the tendency of the inflammatory process to reveal itself by an increase in the body-heat.

Chills. Almost always a feature of the onset, and when absent, supposably, may be slight, or unobserved. They may recur in the course of the disease.

Pulse. Rulably increased, but diagnostically erratic, so far as following the ordinary rules of the relation which should obtain between pulse and temperature. In the early stage, which is one of excitement, it is usually full, active, and greatly accelerated, but even at first, it may be 50 or lower. Toward the termination of a fatal case it may still be low, but usually is high, and may be uncountable. It is liable to great variation within very short periods of time.

Respiration. This is not so much disturbed as one would expect, and, like the pulse, does not follow the temperature. Generally it is quickened in the early stage, and becomes slow and irregular with the advent of coma. Toward the final stage, it is often Cheyne-Stokes in character, although it is a feature of the earlier stages of the Tuberculous form, so that while in most cases it is a sign of death, such is not always the fact. There is another peculiar condition in the course of the disease. The patient is prone to periods where the respiration ceases for several seconds at a time, and, in meningitis of the posterior fossa, there will suddenly occur a failure of breathing with irregular periodicity, cyanosis, and death. Macewen's sign (see page 429).

Nuchal Rigidity. From the first a large proportion of cases will complain of audibly, or by their actions reveal a rigidity of the muscles of the neck. The patient will attempt to get the head back over the pillow, or in the early period of more gradual onset drop the head over the back of a chair, or resist attempts to passively draw the head over toward the chest. It is one of the first unequivocal symptoms, and as the inflammation progresses down the spinal column, it may extend to the muscles of the back in general. It arises from the irritation of the spinal nerve-roots, and is an indication for the determination of the lowest level of the meningitis.

Muscular Rigidity in General. This rigidity is not confined to the neck and back alone, but the board-like rigidity, and even retraction of the abdominal walls is from the same cause in some instances, and in others is a symptom of irritation of the motor cortex of the brain. Tapping the muscles causes the patient to shrink, and percussion of the spinal muscles causes a retraction of the spinal-column. The muscles of the limbs are in a state of irritability, and this is the cause of what is known as Kernig's sign, which is present in about three-fourths of all cases. When this sign is present it is impossible to fully extend the leg so that it is in a straight line with the thigh, if that has been previously placed at a right angle with the trunk.

Test. Place the patient on the edge of the bed, or in a chair, or lying in the bed upon the back. If on the edge of the bed, or in a chair, the thigh will be at right angles with the body, but if in the bed, it must be so placed. Now, with the thigh in this position, try to bring the lower leg in a line with it, and it will be found to become fixed or show very definite resistance at an angle of less than 160 degrees. The arm will at times show the same phenomenon. This is a sign almost universally present, and when found is conclusive evidence that there is irritation of the meninges. At times trismus (lockjaw) will be present, but it is a rare symptom, although this rigidity is more common than a paralysis of the muscles of mastication.

Hyperesthesia. One of the earliest signs is a hyperesthesia of the special senses. This is shown by the desire of patients to be left in a darkened room, and their evident annoyance at the least noise about them. The same is true of the muscles, and is "increased myotatic irritability" referred to under the head of muscular rigidity. The skin is sensitive to pressure, and the tissues of the skull also, since the patient will wince and shrink from percussion. A unilateral hyperesthesia is rare, and hemianesthesia is unknown.

Vasomotor Disturbance. This is an early sign in all cases, and under its influence the skin assumes a dark, and sometimes mottled appearance. If the finger-nail be drawn quickly across the skin, it will be followed by a dark-red discoloration. This is the "tache cerebrale," but it is not diagnostic of meningitis, as such a degree of vasomotor disturbance is seen in many other diseases, but is diagnostic of vasomotor paralysis.

Deep Reflexes. These are usually increased at the outset, but tend to be decreased later in this disease, but sometimes show little perversion. If the patient survives for a week or ten days, they are generally obliterated. There may be a discrepancy upon the two sides of the body. In some cases, there will be sufficient descending degeneration of the motor-tracts, to develope Babinski's toe-sign—extension of the great toe upon stroking the sole of the foot.

Bowels. These are almost universally constipated.

Bladder. The urine is retained as stupor comes on, but occasionally there is incontinence instead, which is the usual condition in the later stages of the disease. The quantity is diminished, as we should expect in the febrile condition, and it is often albuminous, but very rarely does it contain sugar. If the floor of the fourth ventricle is irritated, we may find both albumin and sugar in large amounts.

Cortical Irritation and Softening. Convulsions are common, especially in children, and are usually general and protracted, but they may be upon one side only, or of a single part. The tendency is toward generalization, irrespective of the site of the lesion, since irritation is not so sharply defined, and the whole brain is in a state of excitement. These may occur at any stage, although very common as an initial symptom in severe cases, and may be the only symptom present in septic ones. Beside convulsions, we find twitchings of the muscles, and paralyses of a side, or of a single part; and facial palsy of a cerebral type, i. e., leaving the upper face free, may be a symptom at any stage. Aphasia is common in the tubercular forms, but rare in the others.

Asthenia. A general muscular weakness is always present, and may be quite sharply localized in an intense form in those cases where the process is definitely localized.

Cranial-Nerve Involvement. The cranial nerves are almost always involved, and the facial, oculo-motor, optic, and less often the olfactory suffer to some degree in almost every case, the exudate burrowing along the sheaths of these nerves more universally than in those lower down. Since the facial and the auditory nerves are in contact in the internal auditory canal, they are liable to simultaneous involvement, but deafness is more apt to be caused by degeneration in, or inflammation of the labyrinth than from destruction of the nerve-trunk. The early symptoms of cranial-nerve involvement are, of course, irritative, and hence we find intolerance of light and sound, and occasionally over-sensitiveness to odors. Later these symptoms are replaced by symptoms of destruction, or serious impairment of these nerves, and, therefore, we find loss of function.

Optic Neuritis. This is a notable, but not a constant symptom, rarely complete enough to greatly injure the sight, and common in cases affecting the base, while it is rare in those of the convexity. It rarely goes on to choked disc, and when so it is usually one-sided.

Pupillary Changes. They occur early, and at first the condition is one of contraction, which shows this peculiarity, viz., that one pupil will be contracted more than the other on one day, and the other will be the seat of greater contraction upon another day. At a later period the preliminary contraction is replaced by relaxation, and the pupil is dilated, and the reflex contraction to the stimulus of light is greatly reduced, or altogether wanting. The reaction to cutaneous stimulation (obtained by pinching the skin over the fourth or lowest cervical sympathetic ganglion, which lies just over the neck of the first rib) may be present.

Oculo-motor or Third Nerve. This is very frequently affected in some of its functions, so that the subjective symptoms of diplopia, and its objective and causative symptom, squint, are very common. It is practically always present, and if latent may be developed by having the patient turn the eyes in first one, and then in the other direction, when we shall discover a lack of parallelism between the two eyes. When visible, it may be inconstant, or present only on a particular movement. In an early stage, it depends upon the irritable overaction of one muscle or another, but in a later stage it is the result of a paralysis of a muscle.

Nystagmus is less frequent. The structures of the eye may suffer, and we shall find all grades of conjunctivitis, sometimes choroiditis, and once in a while panophthalmitis with a destruction of the eyeball.

Deafness. This may come on in the course of the disease from an extension of the degeneration, or inflammation to the labyrinth; it is very rarely due to the destruction of the auditory nerve-trunk.

Facial Nerve. The paralysis of this nerve is common, and is shown by change in the line of the mouth, or defect in the power of movement. It is peripheral in type when the meningitis affects the base, i. e., involves the whole face, but if the disease is of the convexity only, it will be of the cerebral type, i. e., leaving the upper part of the face in a normal condition, although it may weaken it at the outset.

The Pneumogastric Nerve is often affected, and such injury is no doubt a frequent cause of the disturbance of respiration and heart's action.

The Hypoglossal Nerve is not always immune, as evidenced by the occasional deviation of the tongue.

Eruptions. Urticaria is often present.

Trophic Changes. The nutrition of the skin is at such a low ebb that mild irritants are apt to produce extensive and severe blistering, and even the heat of a hot-water bottle is apt to cause a slough. There is a strong tendency to the developement of bedsores, particularly in the last stage, but it is more characteristic of the subacute than of the acute cases.

COMPLICATIONS. Sinus thrombosis and cerebral abscess are the most frequent complications.

COURSE AND PROGRESS. There are three fairly well-defined stages: invasion, compression, and coma. While a case may develope coma as a primary symptom, or some amount of paralysis, the usual invasion symptoms are those of excitement, showing that the cerebral cortex is in an irritated condition. Such an irritation will give rise to delirium on the one side, and convulsions and muscular contractions on the other. This stage typically lasts about a week, and during this time vomiting, delirium, vertigo, high temperature, muscular rigidities, diffuse headache, and hyperesthesias are the prominent symptoms. The stage of compression also lasts about a week, and here function of all kinds is reduced; the head is retracted, rigidity becomes more marked, and more constant, we find squinting, and other paralyses, incontinence of the sphincters, and a very slight response to the strongest stimuli, since apathy is increasing toward the last stage, which is coma. In this second stage, of compression, irregularity of respiration is very marked, either from the compression directly, or from involvement of the pneumogastric nuclei in the disease process. The last stage, or that of coma, presents deep stupor, loss of the deep reflexes, incontinence of both bowels and bladder, while the abdomen still remains retracted, the pupils dilate, the skin becomes moist, there is a small and rapid pulse, rapid, superficial, or Cheyne-Stokes respiration, and a general paralysis. Death from exhaustion, or in convulsions, generally ensues in from two to three days. Generally speaking, the symptoms of this stage are of general cerebral disease, rather than of any localized lesion. We have just said that practically each stage lasts about a week. If they survive the first week, the case is apt to be one marked by remissions, and may last for weeks, or months, with variable symptoms, but conclusion in death is a vast probability. The possibility of remissions in the most desperate cases must always be kept in mind, but must not be made a basis for an optimistic prognosis. Meningitis in infants, while possibly fatal in a few days to a week (generally true of cases affecting the convexity), is most often a disease of many weeks, and maybe months. Septic meningitis, and that secondary to infectious disease, is usually cerebro-spinal, and rapidly fatal, possibly in forty-eight hours. A meningitis following an abscess may produce death in a few days, whereas primary infection of the meninges, without the secondary process of abscess, may result in an illness which is fatal in a week or two, and in forms secondary to general disease it may run a course of several weeks. Tuberculous meningitis is peculiar in the long duration, and the insidious character of the invasion stage. This is followed by a stage of irritation lasting from three to ten days, and that of coma, which may last from a day or two to a week or two. Statistics show that one-fourth of the cases die in the first week, after the stage of irritation has set in (there may, of course, have been a long invasion stage), one-fourth die in the second week, one-fourth in the third week, and the other one-fourth during the next five weeks. Syphilitic meningitis furnishes the slowest cases, which may have an invasion period of six or eight weeks, and an irritative stage of irregular severity which may last months, or a year or two.

In general one must expect a fatal issue, except in syphilitic cases, which are usually quickly relieved by anti-syphilitic treatment. There has been almost unanimity in the opinion that tuberculous cases never recover.

That there is a slight possibility of cure is assured by the fact that Hare states that Fuerbiger and others have reported the recovery of cases of meningitis in which tubercle bacilli were found in the cerebro-spinal fluid, and he himself states that he has found cases of healed tuberculosis of the meninges in patients dead of advanced pulmonary tuberculosis at the Henry Phipps Institute.

Tubercular Meningitis. May be weeks, and perhaps many months in duration. Recovery is never complete, and the patients remain defectives to a degree, all their lives. The most usual losses are in the functions presided over by the cranial-nerves, and deafness, which tends to develope mutism, is the most common, unless strabismus surpasses it. On account of the cerebritis, which is to some extent a component in almost all cases, there is a diminution of motor or intellectual power in some part, and to a variable extent. The infantile case is almost certainly followed by some degree of internal hydrocephalus with its attendant damage to the functions, and future developement of the brain.

DIAGNOSIS. The diagnosis in some cases is very easy, while in others it may be clouded by the intensity of the disease to which it is secondary, or the injury which has preceded it. In other instances the symptoms are fragmentary, as in the citation previously made, where cases of septic meningitis have been marked by delirium alone. If it is evident that the brain is organically affected, we are led to believe the condition to be acute leptomeningitis by the method of onset, and the manner in which the symptoms are combined, rather than by the presence of some striking phenomena. In the vast majority of cases, the onset is subacute, i. e., not a matter of hours, but of days, or a week or two in the slower forms of the disease. There is vomiting of a persistent and causeless type, a delirium co-existing with headache, and a headache which is not only severe initially, but persists, and is prominent, even when ordinary sensation has been abolished. This is an important point, since as Jenner has pointed out in other diseases, however severe headache may have been as an initial symptom, it fades into the background as the delirium and general prostration become prominent. There is also an unusual relation between temperature and pulse, there are inequality of the pupils, strabismus and choked disc, nystagmus, weakness of the muscles of the face, retraction of the head; retention or incontinence are liable to be the conditions affecting the bladder and the rectum. Convulsions are apt to occur initially, or after the disease has become established. Ready blistering of the skin would be a confirmatory symptom, like the preceding.

Lafarcinarde says that the tuberculous form may occur in infants under two years of age. It may show a hemiplegic or eclamptic form. There is a somnolent form which may be diagnosed by these four symptoms. Progressive somnolence; absence of winking (ocular catalepsy); progressive emaciation, and an instability and irregularity of the pulse at successive examinations.

Squires has found invariably present by the fourth or fifth day of meningitis a rhythmical contraction and dilation of the pupil, when the head is placed between the knees of the examiner, face upward, with the body supported. Grasp the sides of the child's head with each hand, and produce gradual and forcible extension of the head on the spinal column. As the head is brought back in extension, the pupils will commence to dilate simultaneously with the commencement of the extension, and the degree will be measured by the degree of extension. Upon flexion the pupils contract, so that they are well closed by the time that the chin is forcibly brought to the manubrium.

Bridzinski's Sign is a neck reflex sign, said to be constant in meningitis. Fex the head firmly forward, and the thighs and legs become flexed.

Blood-changes in Meningitis in Children. Leucocytosis means pus, and hence suppuration. In the estimation of the morbid character of findings in children it should be remembered that while 5,000,000 red corpuscles in the male, and 4,500,000 in the female, with about 7,500 white is normal in the adult in health, a different condition obtains in infancy. A child at birth shows about 5,749,500 red and 15,000 to 20,000 white cells to the cubic centimeter.

At the age of one year we ought to find about 15,000, and at the age of seven we obtain the adult count of 7,500. The leucocytes are greatly increased after digestion, and at the outset are increased up to 30,000, and do not reach a normal until about five hours after the ingestion of food. If the food has consisted largely of fluids, or much fluid has been taken with it, there will be an apparent decrease of the red corpuscles. Diarrhea, or other wasting diseases, will produce an increase in the count of both red and white. In tuberculous meningitis the red show no change, or are slightly increased, and there is generally no leucocytosis. The eosinophiles are reduced, while the large lymphocytes, and transitional forms are increased. Normally, the eosinophiles are 1-2 to 4% of the total white, and the large lymphocytes and transitional forms are from 4 to 8%. (Cerebro-spinal fluid, see treatment.)

Differential Diagnosis. Typhoid Fever. This, when affecting children, is much like in its onset, and the petechiæ resemble those of the cerebro-spinal form, but they are confined to the abdomen in typhoid, and this disease lacks the herpes labialis. The bowels are loose instead of constipated, swollen instead of retracted, and the initial headache ceases when delirium comes on, or if persistent, does not at any rate increase.

Febriculæ of children have headache and delirium, but they are initial symptoms only. The fever accounts for their presence, and the headache soon passes away, or fails to increase. Convulsions may occur in both these diseases, but they are initial, and are not presented in the same combination with other symptoms. Eye-symptoms especially are lacking, and if they occur at all in febrile disease, are the results, and not incidents, of the acute stage.

Rheumatism. This may produce the rigidity of the neck, but the muscles are sorer, and while there may be considerable fever, the combined symptoms are lacking. This diagnostic error is, however, not a fanciful one, and the indications may be confusing at times. Urinary findings are helpful.

Uremia should always be eliminated before a diagnosis of meningitis is made.

Muscular (nuchal) Rigidity from Other Causes. In other cases we may find this symptom, but it will be accompanied by swollen glands, and evidences of disturbance of abdominal or other organs, or an angina.

Intra-cranial Tumor. Onset very slow. Chronic. Limbs slowly grow weak, optic neuritis is severe, temperature and pulse are undisturbed, or only intermittently so.

Hydrocephaloid of Marshall Hall. This has the symptoms of acute hydrocephalus from a congestive meningitis, but there is the history of, or existing, exhausting disease, the fontanelles are depressed, and there is no nuchal rigidity.

Hysteria. This may simulate, but organic signs are always wanting.

Meningeal Hemorrhage. This is very rare, but at times closely resembles it, since both diseases are subacute in onset, both have conspicuous delirium, and both follow an injury. Fever, however, is lacking and after similar invasion symptoms, the actual invasion is quite likely to be apoplectic.

Meningism or Meningismus. There are a series of symptoms reminding one of meningitis where even Kernig's sign and coma may appear in the course of pneumonia, enteritis, typhoid fever, rheumatism, and many other diseases, and, in fact, in any febrile complaint in children and more rarely in adults, but in which the cerebro-spinal fluid shows no change. These symptoms subside with the fever or the rupture of an ear-drum. They never produce grave pressure symptoms or paralysis, and the symptoms are rather erethistic. Immediate disappearance from the administration of small quantities of morphine is diagnostic, but the spinal fluid should be the diagnostic field.

TREATMENT. In all cases the patients should be placed in a cool room, darkened and removed from all disturbing noise. They should be guarded from all unnecessary visitors.

The bowels must be thoroughly cleared out and kept so. Calomel gr. 1/10 hourly until effective or ½ drop of Croton Oil in emulsion, according to age, are commonly advised.

In the early stages they are benefited by ice-bags to the head and neck, and heat to the feet. Mustard to the feet may give comfort, and also may be of real benefit. Antipyretics often fail to reduce the temperature, and they so often cause dangerous depression, that from all points of view it is better to employ cold or tepid baths or packs, and hot baths will often accomplish the same result while at the same time they are powerful agents in allaying delirium and restlessness.

Warm baths are useful, and Aufrect advises a bath of ten minutes' duration at a temperature of 100°, and as many as eight have been given in one day with benefit. In all cases where meningitis is secondary to some focus of infection all avenues ought to be kept in a sterile condition. The sterilization of the intestinal tract does not seem to be a possibility. Counter-irritation along the spine is not in favor, on account of the liability of sloughing of the skin in this disease, but it has been claimed that a broad fly-blister on the nape of the neck, reaching from one mastoid process to the other, will arouse the patient from a stuporous condition, and also be actually curative.

Hot poultices (and they must be kept hot) to the upper portion of the spine improve the local circulation, and allay pain to some extent. If vomiting is a feature, feeding should be rectal, and in any case, and by any avenue, must be frequent, and in small quantities. Stimulants are not well borne in the stage of excitement. Efforts to relieve the vomiting should be such as will not be troublesome to the patient, since they are usually ineffectual. In the stage of stupor the head should be kept high, and flexion of the neck should be avoided, since it interferes with the easy return of the blood from the head.

In contradiction to this generally accepted dictum some observers have reported good effects from the hyperemic treatment of von Bier. This is accomplished by placing a rubber band about the neck of the patient sufficiently tight to cause a definite congestion of the face. This is retained for one or two hours and then removed, to be replaced for a similar period in an hour or two.

In this stage full feeding has a positive therapeutic effect, and should be carried out by the rectal method, or by the stomach, or nasal-tube. The patient may be able to take only small quantities, but in the aggregate they should receive by mouth, by rectum, or stomach-tube (after washing out the stomach) a large amount of soup, custard, junket, whey, beef-juice, eggs, koumyss, or other easily digestible food.

Applications of Iodoform ointment (Iodoform 2 drams, Ether 2½ oz., Olive oil to make 8 oz.; shave the head, and rub in liberally once a day.—Wilcox), Iodoform ointment 20% (Crede's), a collodial Silver ointment (Dana), Tartrate of Antimony ointment, Mercurial ointment, have all seemed to be of benefit in some cases.

Cures have been reported in septic cases by the use of autogenous vaccines.

Trephining and ventricular puncture have had advocates, but no general endorsement. Lumbar puncture was introduced by Quincke, and has been used with increasing frequency as time has developed its value. The theory is, that by the process we are put in possession of a knowledge of the constitution of the cerebro-spinal fluid, and will be able to diagnose the variety of meningitis with which we are contending, and also have in our hands a means of diminishing the evil effects of the compression of the brain.

The reduction of pressure is certainly demonstrated, and that it does relieve the stupor and delirium. The pressure returns, but as the operation can be many times repeated without injury, it is a valuable method for the relief of this condition. Diagnostically it is extremely valuable, since it shows by the abnormal force of the flow the amount of intracranial pressure, for in health the flow is drop by drop at intervals of one to several seconds. The normal fluid is clear as water, and contains the merest trace of albumin, if any.

The pressure is increased in all varieties of meningitis. In the tubercular form it is clear, but shows flocculi in suspension, and when it coagulates it is as though it massed about a fine central thread, while other forms coagulate in a mass. The discovery of the tubercle bacillus should, however, be the criterion, and failing in the cerebro-spinal fluid, the other fluids should be examined, and this is true also of the search for other organisms, as they are not always in the cerebro-spinal fluid. In other forms of meningitis the fluid is turbid containing flocculi, a little blood, polynuclear leucocytes, pus and microöganisms. The monuclear lymphocytes are most numerous in tuberculous meningitis, while the polymorphonuclear are more common in the other forms. There may be as many as 300 per cc. In meningitis the cerebro-spinal fluid is cloudy, flocculent, and albumin is increased to the extent of 2%, or more. By the microscope we can detect the character of the microörganisms producing the condition.

The technique of lumbar puncture is as follows: place the patient, lying upon the left side, close to the edge of the bed. The back must be sharply curved, by forcing the knees as close to the head as possible, in order to obtain the greatest separation of the vertebræ, and *must be kept in that position, or the needle may be broken.* Sterilize the skin with iodine over the second and third, or third and fourth lumbar vertebræ, which are in a line with the crests of the ilium. Some advise a preliminary incision of the skin to minimize the pain, but I think that this causes as much pain as the insertion of a proper needle, which is a Quincke needle, whose chisel point is far less liable to damage the fibres of the cauda equina than the ordinary aspirating needle, or a trochar. The skin should now be frozen with ethyl chloride, except in comatose patients, and the needle inserted in the intervertebral space, between the second and third, or third and fourth lumbar vertebræ, in the center line, directing it slightly upward, but keeping it *close to the upper border* of the spinous process of the lower of the two vertebræ, as there is a hump on the under surface of the inner end of the upper vertebral spine. The needle should be three inches long, and should be pushed steadily inward and upward until resistance suddenly ceases, which will occur at a depth of 2 c.m. in an infant, up to 6 c.m. in an adult, i. e., 4/5th of an inch, to 2¼th inches. Any flow of fluid faster than drop by drop shows a pathological increase of pressure. Never use suction. One can with safety draw from a dram to two ounces, depending upon the pressure. Vomiting and collapse have occurred, but are unusual unless an uncalled-for amount has been drawn. The patient's head should be kept low for an hour at least, or the foot of the bed should be elevated. Possible bad results are cardio-respiratory disturbance, asphyxia, panting, nausea, vomiting, sweating, cyanosis, collapse, rigors, cramps, transient paraplegia, sensations of heat, or of general depression, elevated or subnormal temperature, fibrillary muscular contraction, relaxation of the sphincters, retention of urine. The most common result is severe headache, which with all the other symptoms is best met by posture, and artificial respiration, and stimulation.

THERAPEUTICS. The treatment must be largely symptomatic, in the sense that no specifics are known, nor drugs approaching such a degree of potency.

Stage of Invasion. Begin with Aconite in a sthenic case, and persist in its use up to the period when signs of compression begin to show themselves, or paralysis becomes evident. All strengths of the remedy have been advised, from doses of 1/10th of a drop of the tincture for an adult, and 1/100th for an infant, all the way up to the highest potencies. There is no darkness of the skin, but the face is red and flushed, pulse quick and strong.

Belladonna is preferable when the excitement is still higher in grade, and symptoms point specifically to an invasion of the cortex. The patient is especially free from depression, there is no darkness of the skin, but the face is red and flushed, and the pulse is quick and strong. Acuteness of onset is diagnostic, and while there is wild cerebral irritation, there is none of the fidgetiness of Apis. Typically, a child may have gone to sleep well, awakens later with hot head, cold body and extremities, cries out and has spasmodic jerking of the limbs.

Gelsemium has been given the precedence, by some, over the two preceding remedies, especially in the place of Aconite, in those cases which have begun with rapid talking, and a quick nervous manner. Especially in the cerebrospinal form, but its value here is only for a very short space of time, as the next stage comes on very rapidly.

Veratrum vir., in tangible doses, was strongly advocated by Hale (drop 1/100th to 1/10th), and alternation with Hyoscyamus and Belladonna is suggested, if the pulse and temperature remain high. Indications are great irregularity of superficial temperature, vomiting, great aggravation of head from raising it, proportionate ease from lying, the least motion causes increase in nausea and vomiting, considerable unsteadiness in the extremities, rigidity of the neck, scowling and pallor, and flushing of the face upon one cheek only.

Bryonia is preferred in the tuberculous form, but it is not here a remedy for the initial stage, yet this is usually so ill-defined that the case is not often truly appreciated until the second stage has been reached, and a proper interpretation of the symptoms points out that effusion has already occurred.

Strychnia has been given very commonly by the Old School, but is now rather generally condemned, and this objection to the drug in physiological doses harmonizes well with our belief that it is homœopathic to certain types of this disease. Dr. J. T. Simonson reports the successful use of this drug in the sixth potency.

Stage of Irritation. Belladonna is advised by some in this stage also. Agaricus is advised in the 3x dilution for delirious cases. Hyoscyamus is very useful also in mental exaltation, and the delirium is active. In the midst of a perfect stupor the patient reacts to the slightest stimulus. There is mania, which is relieved by violently shaking the head. Stramonium in wilder cases in the same dilution.

Bryonia when the temperature falls, and signs of effusion come on with a falling pulse. It is held by some to be the best remedy. Jahr advises it when the meninges, rather than the cortex, are the seat of attack (the opposite of Belladonna). Excessive pain and sensitiveness of the cranium are indications for its use. The pains are sharp and sticking, and there is a constant chewing motion. Pains are aggravated by motion. The face is dark and congested, the lips are parched, drink is taken hurriedly and impatiently, and the child shrieks with pain. The type is a rather asthenic one. and covers all the symptoms clear up to the developement of the petechiæ. The delirium is rather mild, but the pain is excessive. It is especially well indicated after Aconite, and, like it, is of no value after the paralyses have become evident. There must be a livid and flushed face, high temperature, an easy sweat with nausea, desire to vomit, and constipation with *distended* abdomen, and scanty and painful secretion of urine. It is advised in the 2x.

Apis after secondary infection from the exanthemata with immediate signs of effusion, and a markedly scanty urine. The case begins with sharp cries and fidgetiness, and there is a perversion of the senses. In cases where there is a restlessness of one-half of the body, and a hemiplegia later. It follows Bryonia peculiarly well, and is for those cases with evident effusion where Bryonia has failed to relieve. Rhus has similar symptoms, but there is great bodily restlessness, instead of the mental which is found in Arsenic. Strongly recommended.

Zinc may be of use in this stage where symptoms of irritation exceed those of compression. The child awakens in fear, rolls its head and cries out, complains of a sharp, sticking pain at the root of the nose, and in all the branches of the fifth nerve, and is worse from stimulation. Some authors feel that the potency of this remedy is much over-rated.

Cuprum, either metallicum, or arsenicosum, is extremely beneficial in the irritant stage. The child screams loudly, its fists are clenched, convulsions are violent, the face is pale, the lips are blue, and the eyes are oscillating rapidly. One of the best remedies.

Asthenic forms, where there is little reaction, cause us to think of Arsenic, Rhus, Hellebore, and the petechiæ are also indications.

Iodine is clinically well supported, but the symptomatological indications are meagre. Rhus may be used in frequent doses of the lower dilutions.

Stage of Compression. Opium when the patient is comatose, or in stupor with convulsions, with incontinence of urine and feces, and in the last stage with contractions of muscles. Hale advises the use of this drug in the 2x; dose, a grain every two hours. The author has seen good effects in well-marked conditions from the 200th. It is advised for a coma which does not yield to Belladonna or Hyoscyamus.

Hellebore is advised in the last stages with falling temperature. The forehead is wrinkled, the lower jaw drops, pupils are dilated, and do not react to light, there is automatic action of one side, there are complete mental apathy, with no response to touch or pressure, dark sooty nostrils, and a slow pulse with cold sweat. The value of this remedy is very much in doubt.

Actæa racemosa when convulsions have persisted after the active symptoms have subsided.

Iodoform, in the tuberculous form, a tablet of the 2x every two hours, is as good as the use of the ointment.

Kali iod. In tangible doses in the chronic forms. Sulphur is good as in other diseases as an intercurrent.

Mercurius is to be given in the early stages where we would think of Belladonna, but the throat is sore, and the glands are swollen.

Belladonna. In its place Atropia 3d may be given in solution every one or two hours.

EPIDEMIC CEREBRO-SPINAL MENINGITIS. MENINGOCOCCUS MENINGITIS. CEREBRO-SPINAL FEVER. SPOTTED FEVER.

DEFINITION. An infectious, slightly contagious disease of the meninges of the brain and spinal cord, due to their invasion by the meningococcus (diplococcus intracellularis meningitidis of Weischselbaum). It occurs sporadically, and in epidemics, which may, at a given time, extend over a large portion of a country, distributed in a very irregular manner, not attacking a large number of persons in any one locality, but affecting small scattered groups, and not apparently following lines of travel. The constitutional and local symptoms show great variability, and irregularity, and epidemics vary greatly in virulence and distribution.

SEX. The sexes are affected about equally.

AGE. Children from the age of one to ten years furnish the greatest proportion of the cases, yet in some epidemics they have almost entirely escaped. It was formerly assumed to be a fact that persons over the age of thirty were almost immune, but in the epidemic of 1905 many persons in late adult life, even up to the age of sixty, were victims of the disease. While it is peculiarly true that infants are particularly vulnerable there is no adequate explanation for the fact. At that age there are open bony canals in the sphenoid bone between the naso-pharynx and the meninges, and this was considered an adequate explanation, but this hypothesis was disproved by the fact that infection is through the blood, shows by its simultaneous appearance in the cerebral and spinal meninges, and not by direct extension from the naso-pharynx. The probable reason is the greater susceptibility of infants to all infections.

RACE. There is no particular race immunity, nor vulnerability.

CLIMATE AND ENVIRONMENT. While cases of this disease have been observed in every part of the world, a very large proportion of the cases have occurred in the temperate and subtropic zones, and the disease is practically confined to the north temperate zone. The epidemics usually begin in the winter and increase through the spring, and die out as the warm weather comes on. It is most virulent during the spring months of a cold and wet year. As in most epidemics of disease, the victims of poverty and the denizens of unsanitary habitations furnish the greater number of the cases, but the converse of these conditions does not confer immunity. The poor probably suffer more because their crowded quarters favor the production of a greater number of carriers, and afford these carriers the maximum of opportunity for propagating the disease. The soil of a district is not a factor, for it is as frequent upon sandy soils as on the alluvial, and as frequent on the hill-tops as in the valleys.

ETIOLOGY. This disease arises from infection, almost always by the diplococcus intracellularis meningitidis, but occasionally by the pneumococcus and the streptococcus mucosa. It probably gains its entrance through the upper respiratory tract, although some believed that it might also gain entrance through the gastro-intestinal route, but later investigations upon the micro-organisms render it improbable that it can pass uninjured through the acid secretions of the stomach. That the infection entered by the nasal route was at first deduced from the fact that there was generally a preceding influenza, but it was later verified by the discovery that in the early stages of the disease the menigococcus was always present in the upper respiratory tract, in the naso-pharynx especially. In typical form the organism presents two hemi-ellipses, apposed by their flat surfaces, and separated by a linear space from each other. It shows no distinct capsule. In the exudate from lumbar puncture they are often found within the protoplasm of the leucocytes, hence the name "intracellularis." They are usually single, in pairs, or four, or six may be seen aggregated at once, but they are *never in chains*. If allowed to dry they die in two to three days, in a room-temperature they die in twelve hours, and in from two to twelve hours in direct sunlight, while hydrogen peroxide 3% kills them in one minute, and 30% alcohol is fatal to them. In conclusion it may be said that the organism cannot live in the warmth of an ordinary room, in the fresh air, or sunlight, and is at once destroyed by weak antiseptics.

PATHOLOGY. The essential morbid process is a purulent inflammation of the pia-arachnoid (leptomeninges), the arachnoid initially, since the earliest changes are in the superficial portions of the cortex, sparing the sulci. The earliest lesions are a dilatation of the blood vessels of the pia-arachnoid, and an invasion of leucocytes into their walls, and the lumen of the smaller veins; the meninges of the cord show changes of the same kind, and simultaneously. At times there is evidence of a thrombo-phlebitis. As the leucocyte invasion becomes more general, the trabeculæ and septa of the pia-arachnoid disappear. Spaces of various size are formed containing masses of inflammatory cells. The exudate is predominantly of polymorphonuclears, either normal, or degenerated. Lymphocytes are next in quantity, a smaller number of large cells, thought to be mother cells of the pus cells found in them, and to be derived from the connective tissue cells, and lastly red blood cells; eosinophiles are rarely present. The ventricles and choroid plexus are edematous, congested, and infiltrated with leucocytes. The cerebellum is peculiarly so affected, but as a whole the inflammatory changes are largely confined to the meninges, with little extension to the parenchyma of the brain or cord. As the process goes on, the greater number of the cells in the exudate disappear, while lymphoid and plasma cells appear in large numbers, and there is an organization of the exudate. In sub-acute and chronic cases, the pyramidal cells, and the cells of Purkinje in the cerebellum show evidence of degeneration, while in the cord, there is destruction of the myelin sheaths, degeneration of the fibres in the peripheral zone, the ganglion cells may degenerate, as may also the posterior root ganglia, The microscopic lesions of the central nervous system differ little in this disease from those of any other form of suppurative meningitis. The dura is tense, and at times its vessels show considerable injection. Its entire surface is shining, and small hemorrhages may be seen on those portions covering the base of the brain. On opening the dura a considerable amount of fluid, turbid, or purulent, may escape from the subarachnoid space, varying with the severity and acuteness of the infectious process. In some cases a purulent exudate forms in the first twenty-four hours, while in others it will not occur for several days. In fulminating cases there is an intense injection of the meninges over the entire brain, which from the flattening of its gyri seems to be enlarged. In the sulci of the convexity on account of the later invasion of the pia, there is a small quantity of turbid fluid, while at the base, in the vicinity of the hypophysis, there is often a small quantity of pus. In cases of less severity, where death has taken place in from five to ten days, there is a thick yellowish-green gelatinous exudate, most abundant at the base, and in isolated bands and placques along each side of a congested blood vessel in the sulci of the convexity. In some advanced cases there is present a cap of pus of considerable thickness, covering the upper and lateral surfaces of the frontal lobe of the anterior half of the parietal, so localized on account of the distribution of the anterior and middle cerebral artery (Göppert). The exudate is pronounced at the base on account of the more voluminous sub-arachnoid spaces, and the larger meshes of the membranes. In the cord the dura shows similar changes, mostly confined to the posterior aspect, but the exudate is rarely purulent, and owing to the peculiar division of the arachnoid spaces of the cord, the exudate may be so localized that at certain levels lumbar puncture will disclose only a clear fluid. The ventricles show little change in the first few days of the disease. Soon thereafter they become somewhat dilated, and filled with a turbid fluid containing fibrinous flocculi. The ependymal lining becomes lustreless. The choroid plexus, at first hyperemic, later assumes a grayish, dull appearance. The parenchyma of the brain and cord show few lesions apparent to the naked eye, the vessels are injected, the cortex somewhat edematous, there may be punctiform hemorrhages, but distinct areas of softening are rare, while abscess formation is still more rare. In the chronic stage the exudate on the convexity usually disappears in from fifteen to thirty days, and is replaced by a local or general thickening of the meninges. Yellowish white dense and opaque bands are usually seen along the vessels of the convexity, and dense adhesions are more often found at the base. False cysts may be formed containing clear, turbid, or purulent fluid, producing the so-called saculated meningitis. When now the membranes are stripped off, portions of the cortex often are found attached, and all the membranes may be fused together. The process is similar in the cord, but less general and pronounced. In this stage the ventricles are still markedly dilated, the ependyma is thickened and irregular, its lining membrane has partially disappeared, and it is covered with granulations, which are adherent to the underlying brain tissue. Pus is often present in the dependent portions of the posterior and inferior cornua, after it has entirely disappeared from the surface of the brain. Communication with the ventricles may be obliterated, but Göppert says not so frequently as was formerly supposed. The cranial nerves are similarly affected by their arachnoid envelopes; the 2nd, 3rd, 4th, 5th, 6th, 7th, and 8th are most often affected. The Gasserian ganglion is often found imbedded in pus at an early stage of the disease, and the exudate may extend along three branches to the orbit, the otic ganglion, and the sphenopalatine ganglion. The substance of the ganglion does not show suppuration. In protracted cases the peripheral nerves may show degenerative changes.

PATHOGENESIS. According to Kopetsky, the first effect of the growth of the meningococcus in the meninges is the consumption of the carbohydrate in the cerebro-spinal fluid, and the production of lactic acid. The increase of acidity leads to edema of nerve-tissue and of the meninges, and this is further increased by the compression of the nutrient vessels. A change in the composition of the fluid results, which leads to an alteration in its tension, permeability, and consequent stasis. Interference with the metabolism of the cellular elements of the nervous tissue leads to their degeneration, and the production of poisonous alkaloids, chiefly cholin, which in turn act as nerve poisons. At a later stage of nerve disintegration neutral fat is formed. The anemia which results from compression of the centers in the medulla acts as a stimulant to the vasomotor centers, the blood pressure is thus raised, and the anemia is temporarily overcome. The changes in respiratory rhythm are due chiefly to the increase of intracranial and blood pressure. Nerve tissue proper is degenerated by imbibition of the toxins, similar changes result from the action of other microörganisms which have no selective affinity for nervous tissue, and these alterations are as a rule temporary.

CYTOLOGY. Normal cerebro-spinal fluid is clear, limpid, and resembles spring-water. There are 60-80 c.c. in an adult, with a pressure of from 30-50 m.m. of mercury. (Quincke.) It possesses a few cellular elements, usually not more than 2-7 to the c. m., and polymorphonuclears are never found in health. It rarely coagulates spontaneously, and possesses no toxic qualities, but "in vitro" it is bacteriacidal.

The fluid varies greatly according to the type and stage of the disease. Pressure and quantity vary from 40 m.m. in an infant, to 510, perhaps, in a child of 7 years. In the first twenty-four hours it is frequently clear, or a little opalescent. On standing, the supernatant fluid precipitates a yellow, homogeneous, viscid deposit, but the supernatant fluid becomes clarified. Turbidity or clarity have no *prognostic* significance. Netter and Koch say that two simultaneous tests show exact diagnosis, viz., fermentation of sugar, and agglutination by a specific serum. Heiman and Sternhold say that sugar is usually decreased, and it is asserted that the absence of sugar differentiates it from tuberculous meningitis, but this is not proven. Netter says that for the first twenty-four hours the fluid is clear in 75% of the cases, perhaps slightly opalescent, rich in albumen, and poor in cells, and contains an abundance of menigococci. This may persist up to the third day. and the presence of a clear fluid does not necessarily exclude a meningococcic infection of the meninges, and too much stress cannot be laid upon this point. In the first day or two 80% of the cell content is polymorphonuclear with irregular borders, with 10-15% of lymphocytes, somewhat less number of large cells containing pus cells, and supposed to be the mother cells of them. Eosinophiles are practically absent. With the subsidence of the acute stage the number of cellular elements gradually decreases with a clearing of the fluid. The polymorphonuclears decrease, also the large mononuclears, and the lymphocytes increase. The fluid is now like that of the tuberculous form. Eight to ten days after the complete disappearance of all acute symptoms, the fluid may show a large number of lymphocytes, and it cannot be said how long it may take for the fluid to regain its normal condition. The free organisms disappear first, next the meningococcus, then the polymorphonuclears, and the lymphocytes last of all. There may be great variations in this order, and the changes are greatly accelerated by the use of serum. In the fulminating type, death may occur before there is time for these changes in the fluid, and it may remain clear, as in the mild and abortive types, and the diagnosis must be largely clinical. At the height of the disease the fluid is grayish-yellow, turbid, purulent, and may be too thick to run through the needle. In chronic, or posterior basic types, dry taps are frequent on account of adhesions of the membranes, or occlusion of the foramen of Magendie. When it can be obtained the fluid is generally clear, slightly yellow, or grayish, with few cells, and those are almost exclusively small lymphocytes, the meningococcus being rarely found.

COMMUNICABILITY. The investigations of the New York Board of Health have proved that it is slightly contagious and infectious. This latter phase will be first discussed. In view of its feeble viability it is not probable that it goes through a period of incubation in the outside world, but it is probable that it passes through some changes in the body of a temporary host. The striking feature in its life history is the number of unaffected persons who are carriers of the germ in the naso-pharynx. Other parts of the throat may disclose its presence, tonsils, buccal and nasal cavity, etc., but the naso-pharynx is its peculiar habitat, this being true alike of the patient and the carrier. Persons about the patient are almost sure to be carriers, and the spread of an epidemic seems to depend upon their number. In the epidemic in Silesia there were 327 carriers in the immediate vicinity of each patient. Fluegge says that 70% of persons in close proximity to a case become carriers. As a rule, the coccus disappears from the naso-pharynx of healthy persons in three weeks, but they have known to persist after four and seven months. The method of contagion is then the inhalation of the meningococcus from the breath of a patient, or of a carrier, and possibly the contagion is not direct, but the carrier is a necessary host during some change in the coccus. After its reception it is not now believed that it gains direct access to the meninges, but that in some manner it enters the blood-stream, and thus reaches the meninges. Sporadic and epidemic cases do not differ in origin. The evolution of an epidemic is slow, and cases develope from scattered foci.

SMYPTOMATOLOGY. Much of the symptomatology may be easily deduced from the pathological processes just described. It is evident that if the blood-supply to the cortex is cut off, and if there is an invasion of the same by an enormous number of cells, that there will probably be convulsive seizures, and delirium, and that if there is an increase in the cerebro-spinal fluid it will cause headache and coma. The pressure of the congested meninges upon the roots of the cranial and spinal nerves, and the resulting infiltration of their sheaths will naturally produce pain, muscular twitchings, and finally motor paralysis and disturbances of sensation. It is difficult to describe a typical picture of menigococcus meningitis on account of the wide variation in the severity and distribution of the symptoms, and epidemics also vary as widely in their virulence and symptomatology, so that about all that can be positively asserted is that the later cases of any epidemic are invariably less severe. Beside the fairly typical cases which may be called the ordinary, there are the malignant cases, which may be fulminant, or hyperacute; the mild, the abortive, the intermittent, and the posterior-basic.

Adults and older children commonly exhibit what is termed the ordinary type. It may occur sporadically or in epidemics; it has an abrupt and violent onset, after an incubation period of from three to five days. Some cases will have a prodromal period of general malaise, with nausea, loss of appetite, headache, and pains in the back, often initiated, or accompanied by coryza and sore throat. Usually, however, the patient is attacked by the disease when apparently in perfect health. Infants were supposed to be immune to this disease, especially in the sporadic form, but this opinion is found to have been an error, arising from the liability to a wrong diagnosis before the use of lumbar puncture. In infants the insidious type of onset is most common. The onset of these ordinary cases is marked by a severe headache, with vomiting and vertigo; the patient is very much prostrated, he is sensitive to any light, or noise, he is tormented by frequent chills, which are often replaced in children by convulsions. The patient is restless, but in spite of his disturbing symptoms, he still has a clear mind. The temperature and pulse begin to rise, but the symptoms at this time have little diagnostic value, except that with a temperature of about 102°, as a rule (but the temperature may rise to 104° to 106°) the pulse is rather too low relatively. The spleen is enlarged but is not palpable. At this time a remission may occur, but with the recurrence of the symptoms the stiffness of the neck comes on, and is painful. There are pains which follow the spinal nerves, there is an intense hyperesthesia of the surface of the body, at times tonic and clonic spasms of the extremities are seen, and tremor is common, all of which symptoms indicate the involvement of the spinal meninges. Vomiting may be a very constant symptom, and if a continuing one it is of bad omen. This increase of symptoms takes place about the third day, and the headache becomes more severe. The mind soon becomes clouded by delirium, which usually is mild, but is increased as the fever rises, and at night, and may become so violent that the patient must be restrained. The nuchal rigidity has become so pronounced that the attempt to bend the head forward upon the sternum lifts the patient's body from the bed, and the muscles of the limbs and trunk show the same rigidity. The spasticity of the hamstring muscles is the cause of the presence of Kernig's sign, and this tension often gives rise to local pain. The muscles of the abdomen become so rigid that its outline becomes flat, or concave (boat-shaped abdomen). By this time the deep reflexes are exaggerated, but this rule is not unalterable, as in some cases they are only slightly modified during the course of the disease. Finally the spasticity of the muscles of the back may become so great that the head may be so bent back as to lie between the shoulders, and the back is so arched that the body is supported from the head and the buttocks, or the heels even (Opisthotonus). I have seen one case where the flexion was forward (Emprosthotonus). The hyperesthesia of the surface is now so great that stroking the skin, or pressure upon a muscle, will induce such a degree of pain that the patient will scowl, or cry out, in spite of his dulled mental condition. Infants at this stage will have developed three important symptoms, viz., hyperesthesia, rigidity of the neck, and bulging of the anterior fontanelles. This latter condition should always arouse the suspicion of the physician, especially in the presence of diarrhea, for here rulably the fontanelle should be depressed. The Macewen sign is of no use before the fontanelle is closed, and Kernig's sign is of no value under the age of two. The hyperesthesia of the surface in an infant is, at times, the one prominent symptom, and very early also is a loss of the ability to sit up, and also the presence of tremulousness of the hands. The best way to demonstrate the rigidity is to sit the child up, and move its limbs about to show the hyperesthesia, then lay it upon a hard table, and the rigidity is easily demonstrated. Restlessness may be present, but usually infants lie curled up upon the side, are quiet and sleepy unless disturbed, when they utter piercing shrieks. The initial retention and constipation of adults and older children are now liable to be replaced by incontinence, as is the case in other forms of meningitis. In contradistinction to the general view, Herman and Feldstein found that a majority of their patients preserved normal function of the bowels, with good appetite and marked thirst. The urine is increased in quantity, and shows some albumin, but there is rarely any other sign of nephritis. The face becomes pinched and pale, alternating with flushing, which shows the disturbance of the vasomotor system. The pupils are usually equal, and respond sluggishly to light, and at this stage the ophthalmoscope will usually reveal a beginning optic atrophy. If the head is now grasped, and strongly bent forward on to the sternum, the pupils will alternately dilate and contract (Squire's sign), and sometimes this phenomenon is automatic, and is then termed hippus. About the third day the various eruptions, which are a common but not invariable feature of the disease, make their appearance. Herpetic vesicles develope about the lips and nose, lasting a few days, and appearing in successive crops, and constitute a diagnostic feature of this variety of meningitis. In addition there are petechiæ, diffused over the entire body, but more deep and purple in some than in other localities. Urticaria and purpura may also be present. This cutaneous irritability is, with the added element of vasomotor paralysis, the cause of the "tache cerebrale" of Trousseau, which was formerly considered to be diagnostic of meningitis. The trophic disturbance may be sufficient to cause furunculosis, and in severe cases bedsores occur on the sacrum, occiput, and heels. The cranial nerves are affected in almost every case, and few cases escape some degree of strabismus, or deviation of the eyes. This paralysis is less frequent than in the tuberculous cases. The membranes and structures of the eye are apt to be the seat of all grades of inflammation, even up to destruction of the globe.

Chemosis is almost universal, so as to be quite diagnostic of the cerebrospinal type of meningitis. Acute suppurative otitis media acuta occurs at times, but spontaneous rupture of the drum-head is rare. At this stage the tongue is dry and coated, the temperature is variable, and subject to wide fluctuations within short periods, and the pulse varies as widely, but a very slow pulse is less common than in the tuberculous form. Respiration is rapid, but not out of proportion to the temperature. The bowels are usually constipated, but Goeppert reports a large number of cases where diarrhea was a persistent symptom; dysenteric symptoms are not uncommon. The patient usually lies on his side with the knees drawn up.

By this time the delirium has become very pronounced, and varies with the intensity of the fever, and occasionally a patient may suffer for days at a time from hallucinations, or delusions. The mental state may seriously interfere with feeding, and consequently we may have extreme emaciation. Difficulty in swallowing is often seen, and of articulation also. In cases of this degree of severity, the mind is so clouded that if they recover they retain very little recollection of their sufferings. General and partial convulsions are liable to occur at any stage, but in general it may be said that while cranial-nerve palsies are very common, spinal palsies are very rare. Hemiplegia is the most common form of paralysis, and it is seen in severe cases before death, and while a flaccid paralysis may occur, it is still more rare. This phase is common in some epidemics, and absent in others. In place of cranial-nerve palsies we may see root and girdle-pains in the distribution of the spinal nerves. If the case goes on to a fatal issue, the symptoms are usually bulbar, producing paralysis of the heart and lungs: there may be continuous vomiting, and the patient gradually fails, or he may die in convulsions from cortical irritation. On the other hand, the patient may succumb to a complicating pneumonia, arthritis, cystitis, or bedsores. If recovery takes place, it is usually months before the patient becomes normal, and the weakened muscles regain their former strength and dexterity. While this description forms a reasonably accurate picture of an ordinary case, we may come upon a type in the early stages of an epidemic which is termed "Fulminant." Such a case is characterized by an intensity of onset, and a rapidity in its course, which is strikingly different from the type just described. The case may begin with a violent headache, convulsive seizures, and high fever. In other cases it is like an apoplectic attack, the patient passing rapidly into deep coma. There is extreme prostration, with irregular, thready pulse, labored irregular breathing, often Cheyne-Stokes in character, with coldness, and cyanosis of the extremities. The fatal termination usually occurs in from ten to thirty hours after the onset, often before the characteristic symptoms of rigidity have had time to develope. Infants are also subject to this form of attack. There is also the "Hyper-acute" form, which is seen at the height of the epidemic, and which is like the ordinary form, except in the rapidity with which the disease developes. There are abortive forms, which begin with great severity, but never develope into the typical disease.

There is also a "Protracted or Intermittent" form, with a fever curve which is like a tertian malaria. The paroxysms are longer, however, and the period is more irregular. The meningeal symptoms are usually mild, but the patient may die from exhaustion or bedsores. In the last stages of an epidemic we may see what is termed the "Mild" form, which has a slow onset, a fever hardly above 101°, with moderate headache, and a stiffness of the neck so mild as to appear like a torticollis. The course is often not more than a week, and the patient has so slight a degree of discomfort as to be able to attend to his daily work. Epidemics have occurred in which the superficial symptoms are entirely unlike the classical ones which have been described. Dr. Samuel Gordon described an epidemic in Dublin in 1866-7 in which the patients had a blueness and coldness of the skin like cholera, bruises and ecchymoses like scurvy or typhus fever, hemorrhages and eruptions over the whole body, but especially on the lower limbs, which were dark-brown, purple, or black in color, in large or small spots, flat or elevated, and they might become gangrenous; hemorrhages from the mucous surfaces, nose, mouth, stomach, bowels, and kidneys.

MENINGITIS IN INFANCY.

Posterior basic is almost exclusively seen in the first two years of life, and it may appear in both sporadic and epidemic form. It is merely a variety of meningococcus meningitis (Still and Thursfield). It differs from the adult type only in the primary site of invasion, since the convexity shows some grade of inflammation, although the primary and prominent pathological change is a yellow, purulent fibrinous exudate which fills the cisterna magna, and spreads posteriorly along the cord, inward toward the ventricles, and forward along the base of the brain to the tips of the tempero-sphenoidal lobes, the interpeduncular space and the optic chiasm. The ventricles are almost always dilated, and filled with clear or turbid fluid.

Clinical Course. Cases are seldom seen over the age of two, although cases have been seen in children of five years. The onset is sudden, with fever, vomiting, and sometimes a convulsion, and after several hours or days the retraction of the head sets in. With this is more or less opisthotonus. The head may touch the sacrum, so that the chest and abdomen are curved, and the abdomen rigid and retracted.

The extremities are spastic, and either extended or flexed, and if at all relaxed, will at once become rigid upon being touched. The open fontanelles bulge, and in the very young the sutures will separate, and there is dilatation of the veins of the scalp, due to an increase of fluid in the ventricles. The eyes are open and staring, with dilatation of the pupils, and a retraction of the upper lids, giving a staring look. Strabismus is common in the later stages of the disease, and about one-third show some degree of blindness from a central cause, since the discs are usually normal. The patients are apathetic, and lie quietly for hours, are greedy for food, paroxysmal vomiting is common all through the disease, and it may be projectile. The temperature varies greatly, at the onset ranging from 101-103° for several days to a week, and in the later stages it may be only slightly above normal. It may from time to time show wide excursions, and just before death exhibit hyperpyrexia. Emaciation is as common as retraction of the head. Vasomotor paresis is shown by tache cerebrale, and erythemas. Kernig's sign is difficult to get, on account of rigidity, and Babinski's is rarely present. There is usually a slight leucocytosis of from 15,000 to 30,000, in which it resembles tuberculous meningitis. Dry taps are frequent, either from shutting off the foramen of Magendie, or adhesions at the base of the brain shutting off the ventricles and subarachnoid space from the spinal canal. When the fluid can be obtained it is not infrequently sterile.

Complications and Sequellæ. The most frequent complication is hydrocephalus, and it is also to be remembered among the sequellæ, since it may appear long after the meningitis. Inflammation of joints also occurs.

The most prominent sequel of the disease is deafness, which may appear in mild as well as severe cases, is almost always bilateral, and is apt to produce deaf-mutism. Blindness is unusual, but some degree of ocular paralysis producing strabismus is very common. Paralyses of the spinal nerves, or hemiplegias are not as a rule persistent, but most convalescents are unsteady in gait for a long time, and muscular dexterity is quite slowly attained. Most adult patients are mentally dulled for months after their recovery.

Relapse. Relapses are common, and occur under conditions which would be deceptive in other diseases. If the pulse is over 100, even though the temperature has been normal for some time, the occurrence of a relapse is not unlikely, and so long as Kernig's sign persists, the patient is in danger of a relapse. According to Altman, we may confidently expect recovery if the pulse is regular, and of normal frequency, and the temperature has been normal for fourteen days. In one case a patient seemed well for a month; then symptoms of relapse occurred, with severe general symptoms, and in five days the disease was re-established, and followed the lines of the first attack.

Twelve days later he had a second relapse along the same lines, which finally ended the disease with complete recovery, except some stiffness, and difficulty in walking.

DIAGNOSIS. The essential symptoms are a meningitis of an epidemic character, with conjoined cerebral and spinal symptoms, marked by early opisthotonos, and cutaneous eruptions, especially herpes labialis.

Vincent and Bellot have devised the precipitation reaction; add 1 to 2 drops of antimeningococcus serum to a tube of fresh cerebro-spinal fluid, centrifuged for ten minutes to clear it. Then place the tube in an incubator at 52 c. for a few hours. A precipitate will occur if the meningitis is from the meningococcus, and is an indication also that as long as the reaction continues the serum should be used.

DIFFERENTIAL DIAGNOSIS. As a basic fact, lumbar puncture disclosing a spinal fluid under pressure strongly indicates meningitis, and this finding would reduce the differentiation to very narrow bounds, except for the fact that meningitis is a very frequent complication of typhoid, pneumonia, septic conditions, alcoholism, and injuries of the cranium and the spine.

In Tuberculous Meningitis the cerebro-spinal fluid is clear, limpid, high in albumin, in it are a few shreds, or flocculi, when viewed by transmitted light, and after standing a few hours, a slender white column is found extending to the bottom of the tube, and branching out at both top and bottom. The fluid under the microscope is clear, or contains a few polynuclear leucocytes, and probably the tubercle bacilli. In contrast with this, the fluid in Cerebro-spinal Meningitis is turbid, contains meningococcus, and polymorphonuclear leucocytes in abundance, and almost exclusively. A lesion common in post-basic meningitis is hydrocephalus, shown by Macewen's sign, which is an increased resonance on percussion over the parietal bones, compared to the sound from percussing a ripe watermelon; there is also constant opisthotonos, and frequently we find nystagmus. These symptoms are rare in the tuberculous form, but a retracted abdomen, optic neuritis, and bradycardia with low temperature are characteristic. Clinically also they differ in the constantly insidious onset of the tuberculous case, the profound stupor contrasted with the easily aroused patient with Cerebro-spinal Meningitis. The Tuberculous patient lacks the herpes about the mouth, there is diarrhea instead of constipation, or dysenteric stools of the epidemic case, nor does the epidemic patient awaken with a start and grinding of the teeth, which is a common symptom of the tuberculous type. It might primarily be said that the tuberculous case is always secondary to foci which should be capable of discovery.

Typhoid. The two diseases may so closely resemble each other that it is impossible to differentiate them by the symptoms, since they both have enlargement of the spleen, a roseola, and similar meningeal symptoms. Both may have stiffness of the neck, but in typhoid the stiffness does not extend to the trunk. The temperature of typhoid is, however, distinctive in the early stages, there is distention of the abdomen instead of a retraction, and while both may be constipated, the relaxed bowels of typhoid are diarrheic instead of dysenteric. The Widal reaction is helpful, but is not always diagnostic. There is leucocytosis in meningitis, and not in typhoid.

Uremia. Convulsions may occur in uremic coma, there may be vomiting, and neuchal rigidity, but there is no fever, nor herpes, nor hyperesthesia. A careful examination of the urine will be conclusive, as there are no evidences in meningitis of extensive structural changes in the kidneys.

Influenza. The nervous types of influenza may present so many similar symptoms that lumbar puncture alone can be invoked for a diagnosis.

Poliomyelitis. This disease comes on usually more abruptly than meningitis. It is very apt to involve the meninges to some extent, and it may implicate the whole central nervous system, and in young children is exceedingly apt to be ushered in by convulsions. The paralyses of poliomyelitis are from damage to the cell-nuclei in the cord, and perhaps brain, and, therefore, except at the onset, are not marked by the pain which is so conspicuous in the paralyses of meningitis, which are from the meninges, and, therefore, accompanied by root-symptoms. According to Peabody and Draper the cerebro-spinal fluid in poliomyelitis is perfectly clear, at times fine white flecks may be seen in it, the pressure is moderate, there is an increase in fibrin, after the second to the fourth week there is no increase in the globulin contents.

Meningism. It is usually said to be a complication, but some cases originate with little evidence of their cause, and develope typical symptoms of meningitis, with these exceptions: the rigidity of the neck is usually moderate, or may be absent, Kernig's sign is not at all common, mental disturbances are less marked, the bulging of the fontanelles is not as prominent as in meningitis, and the same is true of Macewen's sign. Lumbar puncture shows the absence of the characteristics of true meningitis. Four or five days limits the run of the disease, and this short duration, and the perfection of recovery at times, is the only proof of its true character.

Rheumatism. This would show a swelling of the joints as an initial symptom, there might be rigidity of the neck from resisting attempts at movement which might cause pain in the muscle, and the same might be true of the back, but there is no similarity in the associated symptom.

Hysteria. During an epidemic of cerebro-spinal meningitis, hysterical persons often show a rigidity of the neck, but associated symptoms will show that it is a disease of the mind, and not of the spinal meninges, and the same is true of opisthotonos occurring under the same conditions. On examination we should find, also, that there is no fever, nor paralysis of the cranial nerves.

PROGNOSIS. In general the disease is rapidly fatal (more frequently in infants, than among adults or older children, we see a fatal issue, or a protracted chronic stage), with death in from five to eight days, and more in the period from the second to the fourth. The mortality is 40% to 50%, being as low as 20% in some epidemics, and as high as 80% in others. Pneumococcus cases are fatal on the average in six days, and meningococcus cases in the epidemic form in eleven days; in the sporadic form in fifty-one days, while the tuberculous type is fatal in fourteen days. Fulminant cases may be fatal in two or three days, while ordinary cases may continue two or three weeks, but some cases run a course of eight or nine weeks, with an intermittency in the activity of the symptoms.

Prognosis is bad in cases which indicate a profound toxemia by the early appearance of hemorrhagic spots, great dyspnea, and unconsciousness. Fatal cases will show a coma which does not remit, or which becomes deeper; there is progressive emaciation, and the sphincters are paralyzed; there is profuse perspiration, and neteorism. The temperature may go as high as 108°, or fall to subnormal, and the pulse is small, frequent, and intermittent. In such cases death will occur at the end of the second, or beginning of the third week. There is no disease where certainty of prognosis is more difficult, so far as mere preservation of life is concerned. The most intense cases will sometimes end in recovery, even after the appearance of symptoms which would be considered unequivocally fatal; and, on the other hand, convalescence must be very complete before we can feel certain that it is not merely a very prolonged remission. There is a class of cases which is not fulminant, nor so severe as to seem hopeless, but improvement is only remission, and after repeated improvements and relapses, the patient finally dies of exhaustion. The favorable cases show it from the first by the slightness of the symptoms. There is either no coma, or it is quite incomplete. and the temperature falls, or becomes intermittent, and by the tenth day the restlessness decreases, and the appetite returns. Such a case, however, may have a very protracted convalescence. Complete recovery is more probable in children than in adults. Cases which live may make a perfect recovery, even in the worst types. On account of the contraction of the exudate which has been poured out over the different parts of the brain, the bulb, and the spinal cord, and the occlusion, partial or complete, of the foramina of the ventricles, we should expect some grade of hydrocephalus, some degree of change in the cortex of the brain, and a detrimental infiltration about the cranial and spinal nerves. Hydrocephalus may occur four weeks after apparent convalescence. In adults sequellæ may appear two and a half years after convalescence. From all these causes the patient's recovery is usually very incomplete, and some symptoms may remain for a very long time, or become permanent. There is a life-long tendency to headache, the disposition is often changed, and in many there is a considerable mental reduction. Tinnitus aurium and strabismus are almost universal; less common is blindness from optic neuritis, or optic atrophy, and from changes in the globe of the eye, but rarely from degeneration of the visual centers. The prognosis is bad for recovery. Choked disc and ocular paralysis may recover, even after two years. Deafness is very frequent in the first two weeks of the disease, is less common in a later stage, but some proportion of the cases will be permanently and generally irreparably deaf, which in its turn tends to deaf-mutism. It may become a permanent deficiency, even after abortive types of the disease. If it persists after three months it is usually permanent. Another residual symptom which is especially marked in children is such an amount of ataxia that the patient sways in walking, and from which it may take years to recover. From the spinal invasion we may get paraplegia, or paralyses of the spinal type, long lasting, or even permanent. Usually there will be some muscular weakness, with normal reflexes, but whatever has been the grade of paralysis, recovery is generally complete. The tendency of the hydrocephalus is to permanently compress the brain, and produce secondary atrophy. The symptoms indicating this condition during convalescence are headache, vomiting, attacks of coma, and recurring convulsions. Recovery from this condition is doubtful. In convalescence the patients are very prone to joint disease, pneumonia, phthisis, and pyemias The prognosis will be more doubtful, if concurrently with the meningitis there is a croupous pneumonia, scarlatina, pericarditis, etc.

TREATMENT. For general therapeutics see the preceding article. See Strychnia particularly. The Flexner serum seems to be the most successful means of combatting this disease. Heinman and Feldstein describe the technique of the serum treatment, and the reasons therefore as follows. The meningococcus, unlike diphtheria bacillus and other micro-organisms against which antitoxin sera have been effective, does not produce its pathogenic effect through the effects of soluble toxin secreted by the organism, but this is an endotoxin, i. e., the toxin is freed only by the disintegration of the organism. The trouble with the sera in such cases is to place the sera in contact with the organisms in a state of sufficient concentration. The therapeutic action must be upon the meningococcus itself. Flexner showed that the rapid disintegration of the coccus was due to the action of an intracellular enzyme, which destroyed the cell-body, thus liberating a toxin which was capable of producing inflammatory changes in the body. The serum inhibits the growth of the cocci, and aids in their disintegration without the intervention of leucocytes, which is Bacteriolysis. In the second place it markedly stimulates phagocytosis, i. e., forms Bacteriotropins. In the third place, it possesses in a moderate degree the power of neutralizing the toxins, i. e., forms Anti-Endotoxins. Opinion is divided as to whether its chief efficiency is due to its bacteriacidal or bacteriotrophic power. The serum must be brought into direct contact with the coccus in considerable strength, as the toxins act only on tissues in their immediate vicinity. It cannot be brought in by the blood-stream, from the fact that it speedily becomes diluted, and because it cannot penetrate the choroid plexus.

While substances pass very slowly from the blood into the spinal fluid, the reverse is equally a fact. That means that a concentrated serum in the spinal fluid is so rapidly diffused into the blood that doses must be repeated very frequently, 24 hours is about the limit of efficient action from a dose as long as the cocci can be demonstrated.

Administration. The serum should be introduced from a funnel, not from a syringe as is furnished by some laboratories. Not less than ten minutes should be consumed in the administration of a dose. The funnel should be kept in a warming chamber to ensure that it is at blood-heat. The needle should not be shifted during the whole administration, or we may give an epidural rather than a subarachnoid instillation. In infants ten to 15 c.c., in adults 30 to 40 c.c., are usually well borne. After instillation the head should be depressed, and the buttocks raised for ten minutes, and then the patient kept in bed for twenty-four hours. A physician should watch the patient for fifteen minutes before leaving. Often patients will have vertigo and intense headache for a time, without serious significance, but at other times they will go into serious collapse, with no other classical sign of shock than shallow and irregular respiration. Sophian discovered that a robust man had a fall of blood-pressure of 30 m.m. after the injection of 12 c.c., and following the instillation of only three more, the blood took a further drop of 30 m.m. He has made it a rule to follow blood-pressure, and to stop the instillation if there is a drop of 20 m.m. This may be difficult, but it is well to watch patients, and if signs of collapse come, give artificial respiration for a long time before giving the patient up. Adrenalin may be useful. If a case is suspected give a full dose, as the percentage of recoveries is highest in those treated during the first day. The meningococcus is, it is to be remembered, not always demonstrable in the first twenty-four hours of the disease. In ordinary cases the dose should be repeated every twenty-four hours for three days; the fluid then should show absence of meningococcus and adventitial cells, and the polynuclears have been largely replaced by lymphocytes. If the fluid is not clear, the serum should be given daily until it is so. If there is evidence of relapse, fever, headache, hyperesthesia, and an accentuation of rigidity, lumbar puncture should be done at once, and if the coccus is discovered, the initial treatment should at once be instituted. As a routine measure lumbar puncture should be done every five days up to the time that the fluid is not only clear, but has regained its normal pressure, about 40 m.m. In malignant cases the serum may be used every twelve hours. The intensive treatment has another reason. If a dose or two of the serum had been given, and after a few days had been found to be inadequate, a subsequent injection at that time might induce anaphylaxis. There is little or no danger of the quantity given under these conditions being too great. In infantile cases of great severity the serum has been introduced into the anterior ventricle in the brain, but thus far with little success, and it has also been injected into suppurating joints, here with benefit. Some cases seem refractory to serum treatment, and it is supposed that the remedy will be the use of selective strains of cocci.

The sore throat should be treated by antiseptics, like Peroxide of Hydrogen, 1 % solution. If there is conjunctivitis, boric acid irrigation should be employed. Painful joints should be wrapped in cotton, and the root-pains in general are better relieved by hot bran-bags packed along the spine and limbs, than by cold applications, as has often been advised. Vomiting may sometimes be allayed by the use of hydrochloric acid. For the first two or three days the patients are better without food, but should be allowed plenty of water if they desire it. After this feeding should be begun by the administration of beef-juice, eggs, and milk, in any quantity short of producing indigestion.

CHAPTER X.

INFLAMMATIONS OF THE BRAIN.

ENCEPHALITIS.

DEFINITION. It is an inflammation of the brain substance, arising from some infection secondary to disease, or perhaps injury. The blood-vessels are involved in various ways (hemorrhage, embolism, and less certainly thrombosis), and there is a hyperemic area, with hemorrhage, and cellular infiltration. The process is acute or chronic, and pathologically it is hemorrhagic or suppurative. The acute form is usually suppurative, but may be hemorrhagic, and both the acute and chronic forms may be local or diffuse. The symptomatology of all forms is more dependent upon the location of the process than upon its pathological character.

AGE. It is very common among children, and very uncommon among adults.

ETIOLOGY. That there might be an inflammation of the brain itself was insisted upon by authors from time to time, but it was finally accepted, after Struempell in 1884 demonstrated a definite condition in the brain from which children often suffered, from which paralysis was liable to result, and which he called Polioencephalitis. It is the same pathological condition as that which occurs in the pons, in the gray matter about the aqueduct of Sylvius (Polioencephalitis superior of Wernicke) and a similar acute process in the medulla, acute bulbar palsy (Polioencephalitis inferior).

ACUTE FOCAL HEMORRHAGIC ENCEPHALITIS.

ETIOLOGY. Whether it is focal or diffuse, the agent producing the acute hemorrhagic form may be influenza, typhoid, diphtheria, syphilis, the exanthemata, malaria, or toxic agents of all kinds, including the poisonous metals, gases, and autointoxicants. It has been believed, from the fact that cases have been autopsied without finding any recognizable cause, that at times it may be due to an infective agent peculiar to itself. This is possible, since it is well known that in some spinal infections the known causative organism disappears in a few days from the site of the inflammation of which it is the certain cause. It may come from traumatisms, which have not been complicated by the advent of infection, but that this is a fact cannot certainly be alleged.

PATHOLOGY. The lesions are in both the white and gray matter, but are more common in the gray matter of the mind and hind brain, involving the cranial nerve nuclei, and the cerebral cortex. The lesion is the same, whatever may be the site. The capillaries are distended, and there are numerous small hemorrhages. The whole area is hyperemic, red in color, and there is a degeneration of the whole neuron, with final disintegration, and replacement of sclerotic areas from neuroglia overgrowth, or by numerous small cysts. As the nervous tissue is never replaced, the victim will suffer losses according to the locality of the disease. While meningitis may be a complication, such is not usually the fact, but the cerebro-spinal fluid is generally increased, and the ventricles may be distended; if so, the lobes of the brain will be compressed.

SYMPTOMATOLOGY. The symptoms are those of an acute infection, with sudden onset. There is apt to be vomiting, headache, convulsions, vertigo, drowsiness, with an elevation of temperature. These symptoms may come on at once, or there may be a long period of incubation. There may be a progression of this process, and the symptoms deepen into coma, and death ensue. In such cases optic neuritis may be seen. More often the picture may be incomplete, the coma is only partial, with delirium, Cheyne-Stokes respiration, and rapid pulse. In the slighter cases the general symptoms are less intense, and some grade of paralysis will be the result, depending for its site and extent upon the locality of the process. Sometimes there is an aphasia, with or without convulsions, and we often find reduction in mentality in children who have suffered from encephalitis. Nuclear ophthalmoplegia, seen in syphilis, is due to such a process.

POLIOENCEPHALITIS superior of Wernicke, an ophthalmoplegia, since the local symptoms are confined to the eye-muscles, was described by him in 1881. It begins as an acute inflammatory, hemorrhagic process, in the nuclei of the eye-muscles, and is fatal in from ten to fourteen days. There is an associated paralysis of all the eye-muscles except the sphincter iridis, and the levator palpebrarum. Associated with this are ataxia and vertigo, so that the person appears drunken, and at times a mental state resembling delirium tremens, somnolence, and agitation may occur. The disc may show a variable picture. (Posey and Spiller.)

POLIOENCEPHALITIS inferior is an acute inflammation of the gray matter of the medulla, which gives rise to the symptoms of bulbar palsy, viz., interference with speech, swallowing and respiration, and is more apt than not to be fatal. Like the superior type, it is not always limited to the gray matter, and so gives rise to varying degrees of paralysis and anesthesia in the parts below.

CEREBELLAR type. Here the patient will develope, after the generalized symptoms of invasion, a cerebellar form of ataxia, and also inco-ordination in all the extremities, with weakness in the parts below, on the same side as the lesion. All these types, as pointed out by Wernicke, are the result of a process indistinguishable from poliomyelitis, except in being localized instead of general.

PROGNOSIS. The prognosis for life is favorable in those cases where the initial symptoms are slight, while it is unfavorable if the constitutional symptoms are severe and prolonged, and the coma is deep. Perfect recovery is rare, as the victims are almost certain to be left with some infirmity of motion, speech, or mentality.

DIAGNOSIS. It is most likely to be confused with meningitis or cerebral hemorrhage. Early focal symptoms, increasing in intensity, indicate encephalitis rather than meningitis, while immediate unconsciousness, and paralysis, with a fall in place of a rise in temperature, would lead to a diagnosis of hemorrhage. Somewhat similar cases are seen, which are characterized by an exquisite sensitiveness in the region of the trigeminus. These cases recover, and are probably due to a congestion of the dura. Sometimes we also see the acute serous meningitis of Quincke, and all these symptoms are relieved by lumbar puncture. (Dana.)

TREATMENT. The patient should have ice applied to the head in the most thorough manner. A repeated application of leeches has seemed to benefit some cases. Aconite, Belladonna, Aurum, Veratrum viride, and Opium are indicated in about the order mentioned. After this treatment for the acute attack, the residual condition must be treated as if there had been an apoplexy.

ENCEPHALITIS. ACUTE SUPPURATIVE FORM. ABSCESS.

AGE. One-half of the cases accur between ten and thirty years of age, and it is rare after sixty.

SEX. Males predominate.

ETIOLOGY. The site of suppuration in this disease is found to be swarming with a great variety of microörganisms, among which the streptococcus is most prominent, and pneumococcus is rare. If the original infective center is remote from the brain, the infection is carried to the brain by the arterial system, but if it is about the cranium, it is conveyed by the venous sinuses, which are in a state of inflammation. It follows typhoid, and typhus, and the other infections, and sometimes malaria, erysipelas, and furnucles about the head and neck, and it may arise from oidium albicans, or thrush. (Dana.) Metastatic emboli from purulent foci, like a cellulitis, abscess of liver, pus in the bronchi or lungs, septic peritonitis, periostitis, and osteomyelitis, tuberculous inflammation of the lungs, rarely fetid bronchitis, all may be a cause of abscess. Suppuration of the mucous membrane of the nose, of the sinuses, or a carious condition of these bones, or those of the orbit, or operation here which becomes septic, may cause abscess Otitis media is the most common cause; more than half of the cases of abscess come not from an initial attack, but where the otitis is chronic. There is an intermittent discharge, which suddenly stops, and acute pain in the ear begins, which in turn is soon followed by brain symptoms. The original attack of otitis may date back to childhood. Small veins and lymphatics pass out from the tympanic cavity to the superior petrosal sinus, and similar vessels empty into it from the tempero-sphenoidal lobes of the brain. Identical vessels come from the mastoid region, and from the lateral lobes of the cerebellum to end in the lateral sinus. The channels of infection are hence very evident; therefore diseases of the ear are apt to set up an abscess in the tempero-sphenoidal region, if the caries is in the upper or anterior wall of the cavity; and in the lateral lobes of the cerebellum, if the caries is in the posterior wall, and the mastoid cells. The abscess may be between the dura and the cranium. Abscesses derived from the nasal and neighboring cavities are apt to be in the frontal lobes, the infection being carried in the venous or lymphatic channels, and is probably the source of most of the so-called idiopathic cases. Metastatic abscesses are found in the area of distribution of the middle meningeal artery. Injuries to the cranium are a common cause of suppurative encephalitis, although traumatism to the scalp, meninges, or brain may cause it. It also may cause abscess at some little distance from the cortex, but usually the suppuration is immediately at the site of injury. In some cases an injury long before, and appearing to be only a slight contusion without evident laceration of the scalp, may be the cause of an abscess.

PATHOLOGY. There are two forms of suppurative encephalitis: one a purulent infiltration, with subsequent softening. The other is characterized by the formation of an abscess, which may become encapsulated. The size is usually that of a walnut, but varies from 2/5th of an inch to 3 inches. When an abscess becomes chronic, a membrane surrounding the purulent cavity is organized from the neuroglia tissue, its formation beginning about the fifteenth day. The capsule may be complete; thin at first, it grows thicker, and later may become calcified; it is usually organized by the third or fourth week. If it remains thin it is liable to rupture. Not infrequently the capsule will be incomplete, and fistulous openings will be found, and the formation of pus, thick, green, brown, or fetid, goes on indefinitely. When there is rupture of a thin capsule on the surface of the brain, a purulent meningitis will result, but if into the ventricles, sudden death will ensue. Abscesses are single as a rule, but if metastatic or pyemic they are liable to be multiple, and small in size. In both purulent infiltration and the formation of abscess, the brain undergoes "red softening," which is a preliminary stage, in which disintegrated nerve elements, leucocytes, and microörganisms are found. The leucocytes rapidly increase, surround the blood-vessels, and cover the nerve-tissue, which is in a state of inflammatory edema. Cells and nerve fibres are gradually destroyed, partly by intoxication, and partly mechanically, and replaced by pus. Softening of cerebral tissue is observed, also, in the immediate neighborhood of an encapsulated abscess.

SYMPTOMATOLOGY. In cases of the diffuse type, and in the so-called "delayed" cases of abscess the initial symptoms are generally mental. The person becomes irritable, loses the power of application, sustained attention, and of memory. A patient of my own showed her illness first by sitting idly before her routine task of examination of papers. Headache is a very early symptom, and is usually continuous, night and day, but it may remit to such a degree as to raise hopes of recovery from the assumed bilious condition, which is suggested by the yellow skin, the emaciation, and the evident cachexia, but which are usually the signs of sepsis. The degree of headache, however, is not diagnostic, nor does its degree furnish a basis for differentiation from meningitis, but the localized character of the pain, and its aggravation by the slightest movement, do make it quite diagnostic of suppurative encephalitis, especially if it is accompanied by fever at the outset. In general, the initial symptoms of abscess are those of an infection, and there is some fever, and chills, but during the course of the disease, while it cannot be said that fever is absent, the temperature is usually normal, or subnormal, and shows little variation. If the temperature is elevated, it is usually from a complicating meningitis or phlebitis. The pulse is generally slow, and it may so remain, even if the temperature is elevated. A rapid pulse indicates either sinus phlebitis, or rupture of the abscess into the ventricle. Vomiting is a common symptom, and is practically always present in conditions which produce a rise of temperature, but is confined to the early stage, except in abscesses of the cerebellum, when it is a continuous symptom. In the febrile cases delirium is common, and it is sometimes attended by convulsions.

Ordinarily the combination and evolution of symptoms is far more diagnostic than the presence of any one symptom. The largest proportion of cases are from chronic otitis, and the symptoms may be divided into three definite stages. There is first a violent pain in the ear, which is developed into severe headache. There are chills, rigors, and insomnia; the patient refuses food. In this stage vomiting is common, but it is not continued into the second stage, unless the abscess is in the cerebellum, and the temperature may be somewhat elevated. The ear-discharge, at this point, may be very much lessened, or cease altogether. These prodromal symptoms occupy two or three days, when they pass into the second stage, or that of full developement of the disease. The temperature is now normal or subnormal, and the pulse is slow, at times from thirty to sixty. The most conspicuous symptom is a lethargy, sleepiness, and lassitude, and the whole mental picture is like that which, as stated above, is apt to produce the latent cases. The respiration may become Cheyne-Stokes in rhythm. Unless it is in, or presses upon the motor cortex, convulsions are rare, but as the increasing size of the growth tends to press upon the motor tracts, monoplegias and paraplegias are common. Optic neuritis is not as common as in the last stage, yet may occur, though it is more often single, and upon the side of the tumor, and the pupils may dilate, but at times it is double. The third stage is marked by a stuporous state, developing into deep coma, and the patient may die without regaining consciousness. In this stage there is apt to be a rise of temperature from a complicating meningitis, or sinus phlebitis, or the abscess may rupture into the ventricle with the appearance of convulsions, and sudden death. In this stage optic neuritis is common, and is generally double, and there may be ophthalmoplegia. The blood shows leucocytosis, and an increase of polymorphonuclear cells to above eighty-five per cent.

MacEwen's sign was proposed as a positive evidence of an increase of pressure at a definite point in the cranium, but while indicative of it, it can hardly be considered conclusive. To elicit it, a stethoscope should be placed on a bald portion of the scalp, or on the forehead, as the presence of hair vitiates the result. Percuss the cranium, part by part, with a percussion hammer; the resulting notes vary, an infant giving a low, dull, and flat sound, while an adult returns a high-pitched, and clear note. There is an increase over the tumor or abscess, as the tension at that point has been raised. One authority gives the pitch as similar to that obtained by percussion of a ripe watermelon. Horseley says that there is tenderness to percussion over either a tumor or abscess, and peculiarly it has been found that the bones have been discovered to be thinned out over an abscess, although it was some distance below the cortex.

The localizing or focal symptoms of an abscess follow the same rules as if it were a tumor. An abscess in the frontal lobe will be apt to lack localizing signs, but might produce blindness at a very early period. The common site, viz., the tempero-sphenoidal are silent areas, but if the abscess is large it will produce pressure upon the internal capsule, and also upon the third nerve, or infect it from a complicating meningitis. We should then find paralysis of the third nerve, paralysis of the body on the opposite side of the ear infected, with the face most affected. If the leg is most affected, we should then find also a hemianesthesia from pressure on the internal capsule. In these cases the facial palsy is of the cerebral type, i. e., it spares the upper face. If the facial nerve is directly implicated in the disease process in the auditory canal, the paralysis will be of the peripheral type, i. e., will affect the whole face, and will be on the same side as the abscess. If the abscess is on the left side, it will diagnostically produce word-deafness (MacEwen). Starr adds that there is an association tract lying deep in the temporal lobe, which connects the temporal center for memories of words heard with the center in the occipital lobe of memories of objects and wards seen. Even a moderate growth in the tempero-sphenoidal lobe is practically sure to interrupt this tract, and, therefore, it is diagnostic of this location, if we find that the patient cannot name an object shown to him, nor can he without difficulty recognize the object from the name. In cerebellar abscess the headache is quite definitely located in the occipital region. As the cerebellum forms the roof of the fourth ventricle, and increase in the size from a growth causes pressure upon the pons and medulla, therefore we find cerebellar symptoms, viz., vertigo, staggering gait, vomiting, diplopia, and nystagmus. The knee-jerk will be reduced on the side of the abscess, but it will be exaggerated on the other side, if there is pressure upon the pons. In addition there will be palsies of the cranial nerves, but paralysis of the facial alone has no localizing value, since, as has been said above, the nerve may be directly affected by the disease in the middle ear.

Prognosis. The prognosis is grave in all types of suppurative encephalitis. Some of the diffuse cases recover, particularly those traumatic ones which are promptly operated. Some of the intoxication cases also recover, but the patient is left with some degree of rigidity and paralysis, or mental change. or both. The focal type of abscess never spontaneously recovers, but goes on to certain fatality unless successfully operated. The operative difficulties are very great, since diagnosis is often questionable, and localization at times is impossible, the evacuation of the thick viscid pus is not always feasible, and after successful drainage the abscess may continue to extend. Some cases run such a rapid course that death takes place in a very short time, from the diffusion of the process, or the rupture of the abscess into the ventricle. A case in my own practice, associated with otorrhea and a discharge, which stopped with a marked aggravation of the headache, and all other symptoms of intra-cranial pressure, was at once relieved by the reappearance of the discharge. In these cases resulting from trauma, or old otorrhea, the abscess may remain latent for months, or even years, without any suspicion of the true condition, when suddenly death takes place from the rupture of the abscess, or an acute meningo-encephalitis in the vicinity of the abscess. This is the so-called "latent" form. To the same group belong those cases where the abscess is situated deep in the frontal lobe, or in the posterior portion of the occipital. In such a case there are few symptoms during life, and death is sudden. However, in some cases of the latent type there is a more gradual course; an acute intercurrent meningitis may aggravate the few existing symptoms, which continue with gradual increase until death. In the capsulated form the growth of an abscess is often very slow, but may be rapid. Acute abscess is, on the average, fatal in from five to fourteen days, and rarely is it survived over thirty days, the traumatic cases being the shortest. If the leucocytosis and the percentage of polymorphonuclears are both high, there is an acute infection, and the system is making a good defense; if the polymorphonuclears are very numerous, and the leucocytes are few, there is an acute infection, and a feeble defense, while if the polymorphonuclears are few, and the leucocytes are many, the patient is recovering, and a low count of both indicates a slight infection. "In some cases the capsule is so perfect that the toxins are unable to escape, there is slight infection, and the blood examinations are of no assistance in diagnosis or prognosis." (Sondern.)

DIAGNOSIS. The diagnosis is made without difficulty when there is evidence of the history of an injury to the skull, or an otitis media of long-standing, but in many cases the symptoms are confusing, and a basis for etiology is wanting. In all suspicious cases the ears, eyes, nose, and sinuses should be carefully examined, and it should be especially remembered that unobtrusive diseases of the nasal fossæ, accessory sinuses, and the orbit, are the most common causes of the so-called idiopathic abscesses in the frontal lobes. A free opening in a tympanum is not so much an evidence of otitis as a cause of abscess, as is a minute one, or the slight scar of a former one. A septic otorrhea, or an intermittent one whose cessation has been followed by headache, give the strongest ground for a suspicion of abscess. In the absence of any obvious cause metastasis should be thought of, and the heart, lungs, and other viscera should be carefully examined. There is a strong probability of an abscess if we find a localized tenderness over some portion of the cranium, dull upon percussion, an increase of surface temperature at that point, mental dullness, delirium, optic neuritis, especially if only on one side, yellowish skin, rapid emaciation, a diminution of chlorides in the urine, leucocytosis with a polymorphonuclear percentage of over 85. If there are grounds for belief that there is an abscess, and that it is secondary to an otitis, the site of operation then limits itself to a choice between the tempero-sphenoidal lobes and the lateral lobes of the cerebellum. The tempero-sphenoidal is one of the so-called silent areas of the brain, but it gives a positive indication if we find the person has word-deafness, and also if he has mind-blindness (alexia), since it is evidence that the center of hearing is more or less completely destroyed, or that a deeper injury has cut off the connecting tract between the center for word-memories, and that for visual memories. The negative grounds for belief would be the presence of general cerebral symptoms without local ones, for the cerebellum does give positive signs of its invasion. There will be headache, quite local, in the occipital region, cerebellar gait, vertigo, nystagmus, retraction of the head, and rigidity of the neck, and often cranial nerve symptoms. There may be weakening of the knee-jerk upon the suspected side, while it is exaggerated on the other, but especially there will be assynergia (lack of coördination in movements) adiadochokinesis (lack of ability to rapidly execute complicated movements), and hypotonia.

Differential Diagnosis. Meningitis has a rapid onset, high temperature, 101° to 104°, rapid and variable pulse, almost certain involvement of the cranial nerves, strabismus is early, and rigidity of the neck is almost certain. There are occasional twitchings of muscles, or spasms of limbs, or convulsions. There is hyperesthesia to light, sound, and touch all over the body. All these symptoms are contrary, wanting, or unusual in brain abscess. Lumbar puncture is the best means of differentiation. In meningitis there is leucocytosis, and a large number of microörganisms in the cerebro-spinal fluid, which is opaque, but not in brain abscess. The tuberculous meningitis of children is confusing, since there is rigidity of the neck, focal epilepsy, or paralysis, elevation of temperature, but the spinal fluid is clear, like that of abscess, and unlike that of meningitis generally.

In Otitis the blood will show a leucocytosis of from 8,000 to 10,000, but a rapid rise to 20,000 to 35,000 taken alone means that more than an otitis is present. We have probably either an abscess, or meningitis, and the count is greater, and the rise in number more rapid in meningitis than in abscess.

Sinus Phlebitis, or Thrombosis of the lateral sinuses, produces a higher fever, with spasmodic variations in range, chills, sweating, and especially swelling and tenderness of the mastoid process, and the jugular vein will show a hard tense swelling. If it is in the cavernous sinus, there will be exophthalmos, and swelling of the conjunctiva may be present. In all sinus thromboses there is early choking of the disc, and venous congestion of the scalp.

Hemorrhagic Pachymeningitis is traumatic, but it has a pain which is superficial and local, and is not especially aggravated by motion, and the fever is more constant than in abscess.

Tumor may give rise to identical symptoms, but its course is more uniformly progressive, and the process is far more prolonged than is the case in abscess. Moreover, convulsions, if they do occur, are more frequent and regular than in abscess. Headache in tumor is more progressive, severer, and longer in duration than it is in abscess. Involvement of the cranial nerves, double optic neuritis and atrophy are more usual in tumor, and are exceptional in abscess.

TREATMENT. As soon as a diagnosis and localization can be made, surgery is the only treatment to be considered, and as abscess, sinus thrombosis, and meningitis suppurativa are all fatal, refinement of diagnosis is not as important as in some other conditions. While awaiting a diagnosis, the case may demand counter-irritation, or ice-caps, or sedatives, and sometimes stimulation in periods of collapse. A free action of the bowels often relieves the pain, and should not be neglected. When diagnosis and localization have been made, the abscess should be drained, and Starr has indicated some very useful points in technique. The pus is usually so thick that an aspirating needle is useless for evacuating it. In place of this he advises a blunt instrument devised by Horseley, which may be pushed down into the brain with the minimum of injury to it, the blades are then separated, and the pus wells out. In drainage also, he points out that this will not be ensured by the use of horsehair, or gauze, but only by the employment of a fairly stiff tube. If the abscess is secondary to otitis, operation will be either over the tempero-sphenoidal region, or over the cerebellum.

CHRONIC ENCEPHALITIS.

Chronic inflammation of the brain tissue may be found at all ages. It is found in adults, at times about tumors, it is common in old syphilitic cases, and in meningitis, and as a meningo-encephalitis it is a constant condition in general paresis. As an isolated condition it is rare in adult life, but is not uncommon in infants and children, and since the brain is at this time in the stage of evolution, or primary developement, it gives rise to characteristic results, which are known as Encephalopathies. They may be the result of prenatal condition, of birth accidents, or of disease or accident occurring after birth. Aside from these gross and evident lesions, such conditions of enfeeblement may be produced that the tissues succumb to the strain of life in early childhood, and develope paralyses, epilepsies, or athetoses, or defer such evidences of imperfect nervous stability until adult life, i. e., atrophy, or sclerosis. In uterine life such results have followed the illness of the mother from the acute infections, intoxications, cachexias, or even traumatism, but syphilis of the mother, or transmitted through the mother, or the alcoholism of either parent, have seemed to be the most demonstrable causes. Alcoholism in the parents has been attributed as a cause in forty per cent. The accidents at the time of birth have been prolonged labor, poisoning the child by the respiration of retained gases, and instrumental violence. While the retention of the child in the birth-canal may produce cerebral congestion, and the respiration of carbon dioxide may produce destructive changes in the cortical cells, and while forceps are capable of injuring the cranium, postmortems on such children have often shown that the vascular system was unnaturally fragile from intra-uterine disease. Actual massive hemorrhage does occur, and is the cause of birth-palsies in some cases. In forty-seven autopsies of infants in a Boston Hospital, eleven deaths were found to be directly caused by intra-cranial hemorrhage, and all these cases had a history of instrumental delivery. Cushing has operated some such cases, found clots, and removed them, and some of these children have developed normally. Spontaneous hemorrhage is very possible, usually from the veins of the pia, and sometimes from the meningeal veins, and the sinuses. The subarachnoid veins lie along the anterior and posterior borders of the lambdoid and coronary sutures. The coronary suture corresponds to the Rolandic fissure, and as these bones slide over one another, rupture is possible, especially if the blood-vessels are fragile. A hemorrhage here will produce pressure immediately, which will cause convulsions, tremor, and paralysis, and when the clot organizes it will directly irritate and compress the brain, and compromise its circulation. While these hemorrhages are usually from the veins as they are about to empty into the great longitudinal sinus, they may come also from numerous small vessels distributed over large areas, producing scattered foci of yellow, retracted brain tissue, especially in the motor zone. More frequently the hemorrhage has come from small vessels on the surface of the pia, and from vessels within the substance of the brain and spinal cord, where it may cause syringomyelia. It will be at the base in vertex presentations, and at the vertex in breech cases. Since the brain is not fully blocked out until birth, and the pyramidal tracts are not fully myelinated until two or three months after, it follows that these intra-uterine and birth accidents will produce agenesis, i. e., unfinished children.

Pathology. In the brains of some infants evidences of large hemorrhages have been found, giving rise to such changes as would be expected in the adult, and such children are born hemiplegics. More generally the process must be considered to be a meningo-encephalitis, since the dura is usually found to be closely attached to the cranium, and the pia to the convolutions. The nervous structure of the cortex is disintegrated, and small cysts are disclosed, which must represent the site of old hemorrhages. More often in cases of ante-natal origin we find porencephaly, and microgyria. Porencephaly is the result of an inflammatory process of an ill-defined character, by which the blood-supply of a definite arterial area has been cut off. Softening has ensued, and a cavity has been found, not always single, but generally bilateral. While many such cavities are superficial, they often reach into the lateral ventricle, and destroy every semblance of nervous tissue in a large portion of the motor area, the third frontal, or the temporal lobe. When the frontal lobe is thus affected, the child is usually an idiot. Owing also to another phase of arterial disease, there may be a primary sclerosis of the convolutions; the sulci may not multiply, but leave the convolutions few in number. The convolution is small, too light in color, and on account of the proliferation of the neuroglia, which afterwards contracts, the sulci are too wide, as are also the perivascular spaces. This is microgyria. Hypertrophy may be found instead of atrophy, but always at the expense of the nervous tissue. Primarily perhaps, but much more probably secondarily, there is an inflammation of the lining membrane of the ventricles; an ependymitis. The gray matter in contact with this is destroyed, and the quantity of the cerebro-spinal fluid is increased, a dropsy of the choroid, and the aqueduct of Sylvius is so congested that the intra-cranial pressure is raised. This increase of pressure flattens out the convolutions, and they not only fail to develope, but undergo atrophy.

Chronic encephalitis, arising in whatever manner it may, produces some grade or form of cerebral sclerosis, and while a lesion in an adult brain leads to secondary degeneration in the pyramidal tracts, in the fetus, or in the infant, it leads to arrest of developement, and an atrophy of the portions of the nervous system below the initial lesion. Atrophy and diminution in bulk of the brain and cord are therefore characteristic of the lesions in question. Such children will present paralyses, malformation, arrest of physical and mental developement, visual anomalies, deafness, and speech-defects, most of these cases being capable of being grouped under two definite heads, viz.. Spastic Infantile Hemiplegia, and Little's Disease, or Diplegia.

DISSEMINATED SCLEROSIS.

MULTIPLE SCLEROSIS—MULTIPLE CEREBRO-SPINAL SCLEROSIS—SCLEROSIS EN PLAQUE.

DEFINITION. This is a rather rare degenerative disease, but less so than has been imagined, as it constitutes one-half to two per cent. of all the organic nervous diseases, the higher percentage being in Europe. This is an encephalitis, and due to a generalized inflammation like tabes. Areas of sclerosis are developed, which may be scattered throughout the whole nervous system, most definitely affecting the spinal cord, yet present to a variable degree in all cases in the bulb, and the ganglia of the brain. The result is a spastic paraplegia, complicated by a peculiarly jerky incoördination in the upper parts of the body, and by bulbar and cerebral symptoms. Frequent remission of all symptoms is a characteristic of the disease. Cases are very apt to be atypical, and difficult of recognition in the early stages.

SYMPTOMS OF A TYPICAL CASE. The person has a weakness of the lower limbs, with spasticity, and an increase of the deep reflexes. The upper limbs are similarly affected, but to a less degree. There is an intention tremor of the hands and arms, scanning speech, bitemporal hemianopsia depending upon a nasal hemiatrophy of the optic nerves, probably nystagmus, and possibly diplopia. Mentality is apt to be reduced, diffused sensory symptoms are possible, but there is no disorder of the sphincters, nor is there atrophy.

DIFFERENTIAL DIAGNOSIS. This is from Hysteria, Primary Lateral Sclerosis, Amyotrophic Lateral Sclerosis, Ataxic Paraplegia, Hereditary Ataxia, Pseudo-Sclerosis, Tabes, Encephalitis, General Paresis, Cerebro-spinal Syphilis, Cerebral Tumor, Bulbar Palsy, Toxic Tremor, Paralysis Agitans.

SEX. While there is a wide variation in groups of statistics, it is reasonably certain that there are more males than females, American statistics making it almost two to one, while foreign tables make them about equally affected.

AGE. The vast majority of cases develope between the ages of twenty-five and thirty-five, the inception of the disease is very rare after the age of forty, but cases have been seen in which the disease became evident only after sixty. On the other hand, a case at the age of four months, the mother having the disease, and several juvenile cases are on record.

HEREDITY. Direct heredity is rarely assumed to be the cause, but there is one instance of a female and her child being simultaneously victims of the disease, and White shows a family record where the disease is traceable for three generations, descending through the female line, through immune mothers. He says that if the indications of this table are correct, unaffected females who have afflicted brothers may transmit the disease. There are instances where two or three children in a family have developed the disease. The principal hereditary influence is that expressed as neuropathic taint, and the disease is prone to occur in individuals, some members of whose family, or some of whose ancestors have been insane, epileptic, choreic, or have suffered from some other form of palsy.

RACE. While we cannot ascribe any numerical preponderance to any given race, it is a fact that cases are more numerous among the older civilizations of Europe than in this country, two per cent. in European statistics, as compared with less than one-half of one per cent. in this country. By some this difference is ascribed to our lack of skill in its identification.

ETIOLOGY. The etiology is unknown. All the infections and intoxications have been alleged to be competent causes, as well as exposure, refrigeration, traumatism directly applied to the spine, indirectly to other parts of the body, as well as psychical shock without material injury. Investigation, however, generally casts doubt upon the causal relation of these conditions. Lebrun (quoted by Jelliffe) states that injury or hardship supplies the conditions, and that then infections of many kinds are competent to produce the disease. Marburg, of Vienna, stated much the same idea in this manner. Persons are poisoned by blood poisons, but they are only efficient when by exposure or traumatism the vessels of the nervous system have been injured. The infections are typhoid, malaria, rheumatism, small-pox, and pneumonia, while less often are measles, erysipelas, diphtheria, scarlet-fever, and pertussis. He also gives syphilis, but the potency of this is generally denied, although it produces a cerebro-spinal symptom-complex, which is difficult of differentiation. It is also denied that alcoholism can be demonstrated as a cause. Schupfer ascribes the infantile and juvenile cases to hereditary syphilis. Bullock (Lancet, Oct. 25th, 1913) believes that it is probable that the disease is caused by a definite organism, since he was able to produce paralysis of the limbs in four rabbits by injecting them with the cerebro-spinal fluid from a patient suffering from multiple sclerosis. This claim is not generally accepted.

PATHOLOGY. It may be almost exclusively spinal, or cerebral, in site, but generally it is cerebro-spinal, and the patches of sclerosis, to the number of a hundred or more, will be found to be scattered throughout the whole extent of the cerebro-spinal axis. They are grayish pink, and if old, are harder than the surrounding tissues, and may retract slightly on section. In size they vary from a pin-head to that of a walnut, but whole regions, like the pons and medulla, may be completely atrophied. These spots begin in the neuroglia, and under the pressure of its proliferation the myelin is degenerated, and finally the axis-cylinders are disintegrated, but not universally. If this process impinges upon cell areas, the cells degenerate, lose their dendrites, and there is wasting of the emergent axon, but secondary degeneration does not take place in nerves until the axis-cylinder is affected. The spots consist of connective tissue elements, neuroglia cells, spider cells, and a mass of fine fibres. The process sometimes involves both the cranial and the spinal nerves, but only for a short distance from the cord. In such cases it is pretty uniformly interstitial, and the axis-cylinders are relatively uninjured.

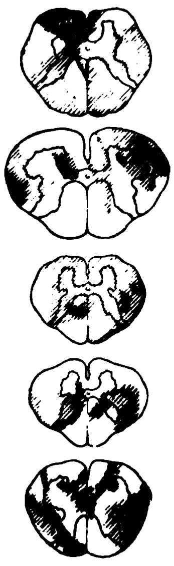


FIG. 77. MULTIPLE SCLEROSIS.
These sections show the variation in the location of lesions at the various levels from the lumbar to the cervical segments.

The process almost universally begins in the white matter, although it often invades the gray; usually, however, as an extension rather than as a primary process. The motor tracts in the cord are the points of election, yet it may invade the posterior columns, giving rise to lesions indistinguishable from those of tabes. From investigation of these sclerotic patches in the formative stage, it has been discovered that an occluded artery is always to be found in the center of such areas. This would indicate that an infection was the basis of the process; that therefrom had arisen a localized thrombosis of a spinal artery, and that the sclerosis was the result of this process. The changes have, therefore, been considered a true inflammatory reaction to an infection acting upon the blood-vessels, but Dana points out that the pathological anatomy does not bear out this view. He says that it is possible that the primary condition is a degeneration affecting first the myelin sheaths, sparing the axis-cylinders and the cell-bodies, this being followed by a neuroglia and connective tissue overgrowth, leading to the formation of small islands of sclerosis. He, therefore, inclines to the view of Bullock (see etiology) that the disease may be due to some protozoan, or other microbe. Complete destruction of any area is rare, since some fibres and cells are always found to be in a normal condition. The greater patches may extend vertically for several segments of the cord; and a vertical extension is most common, but they may, by aggregation, involve the cord transversely to the extent of an entire cross-section. While the distribution may be as widespread or complete as has been stated, the lesions are usually so grouped and distributed that there is little interference with sensory structures, and the motor lesions are so incomplete, from the preservation of some of the motor fibres, that the symptoms are such as would arise from imperfect conduction, rather than such complete losses as would give rise to actual paralysis. Charcot formulated a theory that the characteristic jerky incoordination was the result of the loss of the insulating myelin of the nerve-fibres, perfection of which seems necessary for a perfect transmission of nervous impulses. This explanation is still endorsed by many, but on the other hand, it has been pointed out that when the symptom existed sclerotic patches could always be demonstrated in the pons and medulla, and that bulbar injury was the actual cause of the symptom.

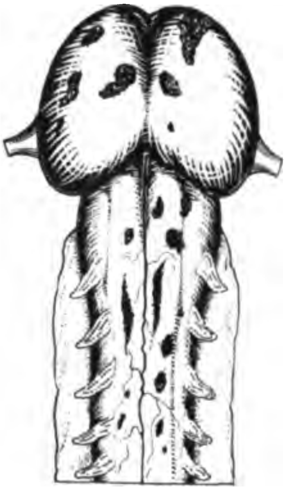


FIGURE 78. Lesions of Multiple Sclerosis in the Cord, Medulla, and Pons.

SYMPTOMATOLOGY. If the fact before mentioned, that the disease may develope to a considerable degree in either the brain or the cord alone, is kept in mind, the atypical character of the symptom-complex will not be so confusing. The typical cases are cerebro-spinal, but a case is none the less disseminated sclerosis, if we find only symptoms dependent upon sclerosis in one only of these areas, and the others need but the passage of time to make their presence evident, since almost all cases finally become typical. Vertigo, speech defect, nystagmus, apoplectiform, and epileptiform seizures, uncalled-for laughter, and tears, mental enfeeblement, and, of very rare occurrence, expansive delusions, are all cerebral in origin. Difficulty in swallowing, paralysis of the vocal cords, atrophy of the tongue, polyuria, glycosuria, attacks of suffocation, ocular palsies and diplopia are from the bulb. A reeling ataxia, as evident with the eyes open as when they are closed, is often from a lesion in the cerebellum. Motor weakness, spasticity, the rare atrophies, disorders of the sphincters and vasomotor irregularities are from lesions in the cord. An appreciation of the point of origin

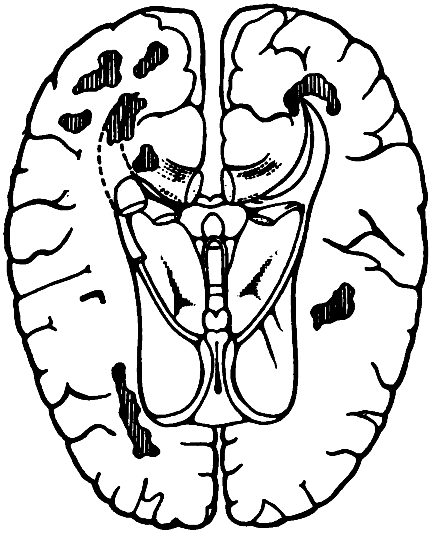


FIGURE 79. Multiple Sclerosis. Lesions in the Brain.

of each symptom will be of material assistance in diagnosis. It is probable that motor weakness is the first symptom in almost every case, but it may be unnoted by the patient, until some striking symptom sends him to the physician, who first is called to attend a patient with an apoplectiform seizure, which is followed by a transient paraplegia, or by some generalized paralysis, which passes away in a few days. This would indicate that the brain was affected at an early stage. Schultze (Deutsche med. Wochenschrift, Mar. 9th, 1911) points out that the temperature is elevated in such attacks, which differentiates them from cerebral hemorrhages.

The most usual initial symptom is weakness of the extremities, lower most frequently, while at the same time the legs are stiff and clumsy. There is in fact some grade of spastic paraplegia. The arms, as has been said, may be the initial point of attack, but much more rarely than the legs, and to whatever extent the case developes, the condition of the arms is usually that of spastic weakness. Diplopia, or weakness of vision, or some form of ocular palsy, sometimes marks the beginning of an attack, and one of my cases, at least, was initiated by sharp pains in the legs. To describe a typical attack, we would say that the patient first suffers from a spastic paraplegia; his gait is very notably disturbed from the weakness of his limbs and the stiffness associated with it; this would naturally make him clumsy in his gait; he has titubation, from the fact that the use of the muscles sets up a spasm in them; there is an ataxia of some form. The variety of this ataxia will be dependent upon some factors not hitherto referred to. If it depends entirely upon a lesion of the motor tracts, it will be a clumsy stumbling gait, the legs are adducted, and crossed over one another. If the cerebellum, on the contrary, is the point of earlier invasion, the ataxia will be cerebellar, which is a peculiarly reeling drunken form. With a coincident lesion of the motor tracts, and of the cerebellar, we shall get a combination which is called a cerebello-spastic ataxia. While not a usual site of sclerosis, the posterior columns are not immune, and, in such a case, we shall get an ataxia like that of tabes, girdle-pains, anesthesias, and sphincter disturbances, all depending upon sensory lesions. The question of implication of the motor tracts can readily be decided by the discovery of weakness and spasticity in the limbs, but between the other two we shall decide that the ataxia is cerebellar if the reeling is as conspicuous with the eyes open as when they are closed, for if it depends upon a lesion of the posterior columns, as in tabes dorsalis, gait and station are very much improved if the patient can correct his movements by the use of his eyes. After the legs have become weakened and spastic, we shall find that on attempts at movement they shake, and sometimes on being touched, which condition is called spinal epilepsy. There is an uncertainty in movement, which is most marked in the upper limbs. When fully developed this is the most conspicuous symptom, and is the well-known intention tremor. Passive, and intention, are the two varieties of tremor. Passive is where the part shakes with some rhythm while at rest, and the part generally grows steady on attempt at motion, and is the form typically seen in paralysis agitans. In the tremor of multiple sclerosis, on the other hand, it is not evident when the part is at rest, but on any attempt at movement it becomes noticeable, and as the object seems on the point of attainment it becomes wider, and wider. It is also aggravated by being the object of attention, or if the patient is emotionally disturbed. It is easily investigated by directing the patient to drink from a glass of water, when he may utterly fail to grasp the glass, or having seized it, he throws its contents about the room in attempting to carry it to his lips. In writing, the pen may be broken by the violence of the movements. Slighter, and less conspicuous incoördination of movement may also be discovered by directing the patient to extend the arms from the body, and with closed eyes to bring together the tips of the extended first fingers, at arm's length, in front of the body. The entire disability results from an interruption of the regular innervation of the muscles requisite for coördinated movement.

The possible variation in the site of the lesion is too great to allow any broad statement of the condition of the reflexes to be entirely true, but where spasticity is predominant, the deep reflexes are exaggerated, and Buzzard states that Babinski's toe-sign has never been absent where it was carefully searched for.

Whenever the posterior columns are affected, the superficial reflexes are abolished.

Nystagmus is an oscillation of the eyeball when it is turned, and fixed in a new direction from a position of repose. It may not show the symptom in more than one direction; either inward, outward, or even upward rotation may be demanded to develope it. Caution should be used in its estimation, since neurasthenic persons may exhibit a false nystagmus if the eyes are forced into *extreme* rotation in any position.

In very frequent cases the oscillation is rotatory. Nystagmus is a symptom of easy determination, and is rare in any other condition, although it has been seen in acute meningitis, rarely in epilepsy, Friedreich's ataxia, cerebellar, and pontile growths, hydrocephalus, tabes dorsalis in an advanced stage, congenital spastic paraplegia, and congenitally in some children, without significance, or known reason. Its presence rests upon the same interference with steady innervation noticed in other parts of the body.

Speech-defect. Late Symptom. This may not be seen, but is distinctive.

It is spoken of as staccato, scanning, or syllabic utterance. The voice may be hoarse. The person is deliberate and monotonous at first, pronouncing the separate syllables in an explosive manner, and becoming hurried, and finally incoherent. With it there may be tremor of the face, tongue and also of the vocal cords. There is a halt also between the words. Since it has been a very pronounced symptom, in the absence of any reasonable post-mortem explanation it has been by some ascribed to cerebral changes, but is probably due to a spastic locking of the muscles, preventing the rapid and smooth readjustment of muscles demanded in speech.

Ophthalmoplegia. Squint is found at times, and any or all of the ocular muscles may be the seat of a paralysis of a transient duration. This symptom is quite uncommon, 17%, and is generally bilateral, and most apparent in combined movements of the eyes, showing that the lesion is in the nuclei.

Optic Atrophy. This is common, and may be preceded by optic neuritis. It has been said to be present in 70% of all cases. From the usual localization of the lesion in the front of the chiasm, the result is almost always a bi-temporal hemianopsia with a white atrophy, of the nasal halves of the optic discs when scoped by the direct method; this will, of course, appear to be of the temporal halves of the discs, if the examination is by the indirect method. The atrophy is distinctively white, instead of the gray variety which is the usual result of implication of this nerve in spinal lesions. The atrophy is sometimes unilateral, but since the macular bundle, the site of the greatest acuity of vision, is almost certain to escape injury, loss of vision is partial, temporary, intermitting, and changing from day to day.

The reduction of vision may be irregular in area, it may be a diminution of the white area alone, and there may be an irregular loss in the appreciation of different colors. When colors are lost, red and green disappear before blue and yellow, which is to some extent diagnostic, since in hysteria the reverse is the rule in color-loss.

The pupils may be contracted and irregularly so; they may react poorly to accommodation, and perhaps to light, but they never actually lose the light reflex, and the Argyll-Robertson pupil (response to accommodation, but not to light), is never seen.

Vertigo. This is a common symptom, and may be an initial one; it is usual at some stage of the disease, but if early in appearance it may disappear later. It arises from defect in parallelism of the eyes, or from a lesion in the brain or bulb. If it is ocular in origin, it may be neutralized by covering one eye, but if it is cerebral this measure will not improve it.

Bulbar Symptoms. The bulb may be affected at an early stage, but usually its symptoms are of late appearance, and often are the cause of death. If the floor of the fourth ventricle is the seat of a patch of sclerosis, and it is a common site, the resulting disorders of respiration and the action of the heart prove quickly fatal. The more general symptoms, which may be tolerated for a time, are like those of bulbar palsy. There is difficulty in swallowing and in mastication. The tongue becomes paralyzed to a variable extent, there may be wasting, and the lips and lower face tremble in speaking. Other results are polyuria, and glycosuria.

Sensory Symptoms. As a rule, sensation is not disturbed, but neuralgic pains in the legs have been noted as initial symptoms at times, and neuralgic pains in the course of the disease are not rare. Hobhouse (Lancet, Feb. 18th, 1905) reported changes of sensation as the initial symptom in thirteen of his sixteen cases.

The sensory changes, as a rule, are subjective, and indicate that the brain and the spinal centers are affected, and take the form of numbness, and tingling, formication, and heat, or a full sensation in the limbs. Gastric crises, girdle- and lightning-pains are possible, but much more rare of occurrence, and indicate a lesion of the posterior columns, or roots. With a lesion in these areas the appreciation of touch, heat, cold, and pain may be diminished to any degree. Headache is common, but intermittent.

Special Senses. There is often a diminution. Vision frequently fails, as has been said, and smell and taste, but deafness is quite rare.

Mental Changes. The intellect is almost always reduced in acuity, and rarely the patient is the subject of expansive delusions. There is a similarity between the physical and the mental losses. Muscular coördination requires the simultaneous action of many muscles to insure a steady and well-proportioned muscular result. In the same manner, a mental concept is the result of an extensive coördination of separate parts of the brain, and the presence of spots of sclerosis in the conduction-tracts, i. e., the white substance, which is their initial site, is fatal to correct mental effort. The mind may be reduced to dementia. Emotionalism, shown in involuntary tears, or laughter, is common, and indicates an invasion of the basal ganglia by the sclerotic process, which we know is a common occurrence.

Among the more uncommon effects, we sometimes see slight visceral changes, and failures in nutrition. The muscles may waste to some extent, but they never show the complete reaction of degeneration. If the functions of the bladder and rectum are disturbed, it is usually slightly and transiently. Very rarely cases have been seen where the process had such a complete transverse distribution that the symptoms of a myelitis were present, and in this case there were rectal and vesical incontinence, the walls of the bladder were the seat of atrophic changes, and a succeeding ulceration, and bedsores developed.

Another symptom which may occur is contracture. The motor tracts are subject to degeneration, and in this case we shall find contractures occurring in from six to eight years. When this stage is reached we find the limbs spastic, with increased deep reflexes, and contracted, as in congenital spastic paraplegia, in strong adduction.

COURSE. Owing to difference in symptoms between a cerebral and a spinal form of invasion, we can see the reason for the wide variation in duration of the different cases. The limit of duration is from two to twenty years, and the vast proportion of the cases last from five to fifteen years. Remission at any stage, and for a period from days to months, or even years, is a prominent characteristic of the disease. We may, with some advantage, consider it as divided into three stages. The first, of invasion, where we find spastic paralysis of the limbs, with titubation, or ataxia, or both, with vertigo, and perhaps also pains in the head and legs. Convulsions, or apoplectiform attacks may usher in the disease, or occur later. In the second stage all these symptoms increase, and added to them are scanning speech, nystagmus, and intention tremor; defects of vision and ocular control are present by this time. Spinal epilepsy is liable now to occur, and late in this stage we may see contracture. The third stage is entered upon when appetite begins to fail, the patient emaciates, paralysis becomes so great, when the spasticity is also considered, that locomotion is impossible, and the upper limbs are helpless. The functions of the bladder and rectum are disturbed, and bedsores appear, and death closes the scene, either from a general inefficiency of the functions, or from some intercurrent affection which the weakened organism could not resist.

PROGNOSIS. If the brain is the seat of the disease, or the bulb is speedily implicated, the patient will probably die in two years, or less. In the purely spinal cases there is no immediate danger to life. Taken as a whole the cases tend only slightly to a fatal issue, except from deficient vitality. Remissions are the rule, and may be long-lasting. Cases have been known to recover.

DIAGNOSIS. Many cases are atypical, and only by exclusion can we arrive at a diagnosis. Taylor (Journal of Nervous and Mental Diseases, June, 1906) rightly says, "The diagnosis is often absolutely impossible to make with assurance during life, even in the advanced stages of the disease. The disease may run its entire course without the appearance of the so-called cardinal symptoms; in the early stages they are very frequently absent, and the question of other, and subordinate signs becomes of increasing importance." Speech defect and nystagmus are not so often present as the text-books have indicated. However, if we find an uncomplicated spastic paraplegia in an adult without the signs or history of a previous cerebral lesion to explain its presence, there is a strong probability that it is the early stage of a multiple sclerosis, especially if it is in a person below the age of thirty-five.

DIFFERENTIAL DIAGNOSIS. Hysteria. This may be antecedent to or combined with multiple sclerosis, and the whole symptom complex, its genesis and method of evolution, must be taken into account, or mistakes will be made. Hysteria is always the result of psychic influences upon a neurotic temperament, and it is possible to dispel all symptoms by a similar shock. Tremor, difficulties of speech, incoördination, paralyses and contractures, may all be dispelled by a command, or a sudden appeal to the emotions. Complete paralysis of one part, with a normal condition of the corresponding limb on the other side of the body, is always hysterical. Tremor of all kinds, and of all parts, may be hysterical, but it is smaller in range, and accompanied by a tardiness of muscular effort, as if the patient wished to exert some degree of force. It is not so closely associated with voluntary effort, is more irregular and variable than in multiple sclerosis. Scanning speech is a possible symptom of hysteria, but is variable in amount in a single sentence, and, if present, lasts through all stages of the attack while in multiple sclerosis it is a late symptom. Color-loss is common in hysteria, but when it occurs, blue and yellow are first lost, while in multiple sclerosis red and green are the first to disappear. These are the symptoms impossible in hysteria: nystagmus never occurs, nor optic atrophy, nor loss of sphincter control, nor the oscillation of the head and body which are so common in multiple sclerosis. Hysteria is not possible, if when the knee-jerk is exaggerated the plantar reflex is abolished.

Primary Lateral Sclerosis. While the gait is stiff and paretic, the spasticity is not so great as in multiple sclerosis; it has not the jerkiness of gait so characteristic of multiple sclerosis. The spasticity, and exaggeration of the deep reflexes are more marked in lateral sclerosis, but in this latter disease sensation is not disturbed at all, and the control of the sphincter is perfect, except in the most unusual cases. Lateral sclerosis displays more atrophy. In rare cases, where the sclerotic patches are located in the dorsal region, and a secondary degeneration has occurred below it, we must rely upon the symptoms in the upper part of the body to differentiate. Lateral sclerosis has no ataxia, intention tremor, nystagmus, scanning speech, nor cerebral symptoms.

In Infantile Spastic Paraplegia, or the congenital form known as Little's Disease, when it is paraplegic, the history will generally be a guide, but, if that is lacking, the presence of cross-legged progression on the one hand, and the absence of distinctive multiple sclerosis symptoms on the other, will render a mistake improbable. When infantile or congenital spinal paralysis is quadriplegic there is a mental defect, but the other distinctive symptoms are still lacking. In no case, except in multiple sclerosis, does the condition remit.

Amyotrophic Lateral Sclerosis. It may be mistaken for multiple sclerosis, but atrophy is more marked, spasticity is less, and sensory symptoms are almost always absent; the characteristic symptoms in the upper part of the body are lacking, and there are no remissions.

Ataxic Paraplegia. While there may be a similarity in the condition of the lower limbs, the progress of ataxic paraplegia is steady, and it shows no intention tremor, scanning speech, nor disturbance of the brain.

Hereditary Ataxia. The diagnosis is very difficult if some of the family have multiple sclerosis, which is apt enough to be the fact for it to be considered. It is as unusual for multiple sclerosis to develope before the twentieth year as to have the hereditary ataxias develope after that period (Marie's may do so). There is a long history of cases in the family of hereditary ataxia. Friedreich's, in particular, developes in early adolescense; it has motor difficulties, but they are pure ataxias, never spastic, and the limbs are flaccid; the deep reflexes are lost, and there is no optic atrophy. Marie's type presents more difficulties, since it developes in late youth, has ataxia, nystagmus, tremor of the head, body, and limbs. mental failure, and optic atrophy is possible. The deep reflexes are also exaggerated, but there is never an ankle-clonus. The motor difficulty here also is ataxic, and not spastic, more of a cerebellar type, while in multiple sclerosis the ataxia is always attended by some spasticity. Apoplectiform seizures are not seen in the hereditary ataxias, nor do we find remissions. The speech is defective, but it is not syllabic, and while there are choreiform jerkings of the arms on voluntary movement, it is not an intention tremor, although similar to it.

Pseudo-Sclerosis of Westpfal. This may reveal identical symptoms on examination, but there are differences on careful investigation, and the history of the evolution of the case is different. Psychic symptoms appear early, apathy even to dementia, and delirium. There is slowness in all movements, there is an absence of nystagmus, and the paradoxical contraction is present. This is the contraction of an extensor, like the tibialis anticus, when a flexor is put on the stretch, as when attempting to elicit ankle-clonus. It is elsewhere found only in paralysis agitans and the neuroses. There is a slow tremor, 2 to 3 per second, and it may be passive. Optic atrophy is not present. Post-mortem, no lesions have been found, so that it has been considered a neurosis. It is a very rare disease.

Tabes Dorsalis. This may be said to differ too vitally to be often the subject of mistake, but, since both have ataxia and incoördination, it is well to point out that the ataxia of multiple sclerosis is motor and spastic, while that of tabes is sensory and flaccid. The deep reflexes are exaggerated in multiple sclerosis, diminished early, and finally lost in tabes. Knee-jerk may be lost also in multiple sclerosis, but loss is transient. Permanent in tabes. Romberg is marked in tabes, but while the multiple sclerosis patient may sway in station, the amount is not increased by closing the eyes. The tabetic is incoördinate, but on attempting a movement he does not break out into such a wild range of jerky muscular contractions. Degenerative symptoms are common in tabes, rare in multiple sclerosis. Fulgurating pains, anesthesias, and analgesias are rarely found in multiple sclerosis, and are usual in tabes. Both diseases are marked by frequent and long remissions.

Encephalitis, Meningo-Encephalitis, and Multiple Softening. There is a form of meningo-encephalitis which may end in multiple sclerosis. It is very difficult to detect any differences, but in some cases we can make a differentiation from the fact that the mental state is one of irritability, restlessness, and excitement, instead of reduction only, that the tremor in writing is finer, and the incoördination is of a tremulous instead of a violent type. Speech is also tremulous, and there is fibrillation of the tongue. If the disease opens with apoplectiform seizures the prognosis must be guarded, since it may indicate encephalitis, beginning sclerosis, or paresis, but in the absence of alcoholism, syphilis, or a hypertrophied heart, such an attack with a transient paralysis is strongly suggestive of beginning sclerosis.

General Paresis. Intermittency of symptoms, and an irregularity of course suggest multiple sclerosis, while slurring speech, Argyll-Robertson pupils, with loss of knee-jerk (although it may be temporarily exaggerated), with the presence of grandiose delusions, or delirium, indicate paresis.

Cerebral Tumor. In general it may be said that both multiple sclerosis and cerebral tumor have tremor, optic neuritis, headache, exaggerated reflexes, and weakness of extremities, but they differ first in the fact that in tumor progress is steady without remission. The head is not painful to percussion in multiple sclerosis, but may be in tumor. If optic neuritis is present in multiple sclerosis, it rarely reaches the stage of choked disc, it is generally partial, usually affects only one eye, and is often transient. In tumor, also, headache is generally constant, and severe, while it is incidental in multiple sclerosis. The vomiting, stupor, and retardation of the pulse, which are common in tumor, are not seen in multiple sclerosis. Both diseases have tremor, but that of tumor is rapid oscillation, and it is not intention in character. Cerebral tumor may give rise to an ataxia resembling that of multiple sclerosis. A tumor in the pons, or the crus, may cause a very similar jerky incoördination, but it lacks the remission of multiple sclerosis.

Bulbar Palsy. Bulbar palsy has a speech defect, and there may be a loss of power of articulation, and of deglutition, and apoplectic seizures may occur. Cases have been seen where there has been spasticity of the lower limbs from irritation of the descending motor tracts as they pass through the bulb. This spastic paraplegia is, however, unusual, and only partial at the most, while in multiple sclerosis it is a permanent feature, and an early one, except in those cases which are bulbar in primary localization. These cases differ from bulbar palsy in the fact that when the lesion is so localized the result is not the wasted tongue, and trembling lips, and general atrophy and immobility of the lower face, but is tachycardia, dysarthria, instead of a slurring speech, and embarrassed respiration. In bulbar palsy, also, the laryngeal reflex is lost, but is not in multiple sclerosis; the mind is clear, but is disturbed in multiple sclerosis. Termination in death by fault of deglutition, bronchitis, or broncho-pneumonia in three or four years is the rule in bulbar palsy, while in multiple sclerosis life may be prolonged for twenty years. In particular, multiple sclerosis is marked by frequent remissions and intermissions, whereas bulbar palsy is a disease of steady progression, or at least there is no remission.

Toxic Tremor. This is said never to be of the intention variety, but that is not a sufficient point of differentiation. In any disease characterized by tremor, differentiation from multiple sclerosis must be made by the course of the disease, by the presence of symptoms not found in multiple sclerosis, and by the absence of symptoms which are found in that disease. All toxic tremors have associated symptoms which are peculiar to themselves, and are diagnostic.

Paralysis Agitans. This disease has tremor, rigidity, muscular weakness, and a changed mental state, often emotional, but its tremor is very rarely intention. There is a rigidity of the whole body, which is from changes in the muscles, and not from spasticity; for a while the reflexes may be exaggerated, but generally are normal. Sensation too is disturbed toward hyperesthesia, while in multiple sclerosis it is usually toward anesthesia. The trophic symptom of "glossy skin" is common in paralysis agitans, and uncommon in multiple sclerosis. Vaso-motor disturbances are notably frequent in paralysis agitans, and rare in multiple sclerosis. The face is expressionless without tremor, but if the face is affected in multiple sclerosis it produces a tremor. The voice in paralysis agitans is high pitched, and speech is hesitating and monotonous, not scanning, nor staccato as in multiple sclerosis. The tremor in paralysis agitans is always regular, however wide it may be, and it is very uniformly located in the upper extremities, but the character of the tremor alone is not enough to differentiate the two diseases. Paralysis agitans is a disease occurring between fifty and sixty years of age, extremely rare in early life, never before puberty.

TREATMENT. This is not always futile for relief, and ought to be always kept in progress, since certain cases cease to advance, and our care may have aided in producing this cessation. Three cases of the author's have apparently been benefitted by medication, both symptomatically, and by a more or less pronounced remission of the whole condition, under conditions giving some grounds for a comparison of methods. The best measures are rest, forced feeding, massage; hydrotherapy may do good, but *hot* baths are to be avoided. Electricity is a matter of difference of opinion, but is advised by some. Quiet life without over-exertion. *Do not* put the patient to bed if it can be avoided, since it has been found that a few days' rest in bed may make a hopeless paralytic of a patient who previously had fair powers of locomotion.

Ataxia, if it occurs, is susceptible to re-education, when a remission has been established. Rigidities may be relieved by counter-irritation of the back, and Swedish movements. Severe pain and contractures have been relieved by section of the spinal roots, but it must be remembered that severe atrophies may result, and cases have been known to die from bed-sores thus produced. Injection of the nerves with 50% alcohol is much safer, and is generally effective.

Electrical Treatment. It is generally believed that this disease is not favorably influenced by electrical treatment of any kind. The disease is an interstitial overgrowth, and not cellular. Growth of this kind is not as amenable to treatment as when the lesion affects other kinds of tissue. It must, however, be remembered that this is a disease which is not in itself fatal to life, and despondency, and resultant resistance to the ordinary diseases of mankind is very much lessened if the patient feels that an effort is being made to remedy his condition. On that account, therefore, I feel that he should have treatment of such a tangible kind, and one which meets so fully the desires of his friends. The most reasonable plan has been stated by King. He says that the current should be given in the direction of conduction of the tracts, and that, therefore, when the posterior columns are the seat of the trouble that the current should be an ascending one, and if the antero-lateral, it should be descending. If, however, the disease be most marked in the anterior horns, the current should be made to pass transversely through the diseased segments. He says also that the greatest effect will be in strengthening the muscles by giving them active exercise with the interrupted galvanic current, application being made over the motor points, or still better than the interrupted, is the use of the sinusoidal. Galvanism mitigates the tremor but slightly, if at all. If muscular contraction can be produced by static sparks the effect is similar, but slighter than from the galvanic. Theoretically the tremor should be helped by giving stabile anodal applications over the diseased nerve-trunks.

THERAPEUTICS. No therapeutic measures promise a large measure of relief, on account of the character of the lesions, but many cases remit, and some seem to recover; so that we have grounds for belief that the number might be increased if the treatment was correct, and symptomatology furnishes the most promising basis, in our present state of ignorance of the actual cause of the disease. The remedies which have seemed most apt are the following, and I have usually employed them in the 6th potency. Aurum mur. when the posterior columns are also affected, shown by the presence of sensory symptoms. Argentum nit. and Mercurius sol. for trembling with paralytic weakness. Baryta carb. and mur. both have paralytic weakness with tremor. Belladonna as above with diplopia and dimness of vision. Calcarea for constitutional degeneration. Causticum, paralysis with intact sensibility. Conium, weak, trembling gait with reeling (implication of the direct cerebellar tracts, or cerebellum). Crotalus, paralysis with no sensory symptoms. Cuprum, paralysis with spasticity. Lathyrus, paralysis with ataxia, and rarely a disturbance of sensibility. Lycopodium, paralysis with formication. Mercurius, general tendency to neuroglia overgrowth. Natrum mur., paralysis with spasticity. Nux vomica, spastic paraplegia. Oxalic acid, paralysis with pains and bulbar symptoms. Phosphorus, paralytic weakness and ataxia. Physostigma, paralysis with ataxia and muscular cramps. Picric acid, weakness of the limbs, with spasms and priapism. Plumbum, tremors and paralysis with cramps in muscles. Rhododendron, paralytic weakness with formication. Silicea, when the posterior columns are affected. Tarantula, with spasms in muscles. Thuja, paralysis with tremor.

Physiological medicine inclines toward the use of Arsenic, and Mercury, while Strychnine in full doses is thought to have a good effect in all except spastic cases. I have seen great benefit follow the administration of large doses of Iodide of Potassium.

THE CEREBRAL PALSIES OF CHILDREN.

A convenient form for graphically presenting the subject is Sach's table.

GROUPS.	LESIONS.
Paralysis of Intra-uterine onset.	Large cerebral defects. Porencephaly. Defective developement of the pyramidal tracts. Cortical Agenesis, highest nerve-elements affected.
Birth-palsies.	Meningeal hemorrhage, rarely intra-cerebral. Later conditions are Chronic Meningo-encephalitis, Sclerosis, Cysts and partial Atrophies.
Acquired Palsies.	Hemorrhage, meningeal and rarely cerebral. Thrombosis from endarteritis and Marantic conditions. Embolism. Resulting conditions are: Atrophy, Cysts, and diffuse and lobar Sclerosis. Chronic meningitis. Hydrocephalus rarely arises from this. Primary encephalitis (the Polio-encephalitis corticalis acuta of Struempel?).

SYMPTOMATOLOGY. The characteristic loss in the congenital and birth palsies is diplegia, and paraplegia, while hemiplegia is rare. When it is congenital and paraplegic, it is the typical Little's Disease. Birth palsies are now rather generally grouped under this caption, but Little, who first accurately described this condition in 1862, demands the fulfilment of three conditions for its identification, viz., it must be congenital; cerebral in origin; and the symptoms must be of a kind as would arise from a lack of developement of the pyramidal tracts. There is evidence of lack of intra-uterine developement, to an extent that we must term it an unfinished child. They commonly weigh less than four pounds, and the birth is generally premature. Hence there is good ground for considering the symptoms at birth as results of imperfect fitness for life, rather than of injury at the time. In these cases there may be almost every combination of paralysis, except a distinct paralysis of the face. At birth the congenital case may be found to be incapable of motion in the lower limbs, and upper as well, perhaps, but there may be no obtrusive sign of any paralysis for weeks or months. The common phenomenon in these cases is a prolonged period of asphyxiation, and a difficulty in swallowing, while convulsions are quite rare (contrary to the rule in birth-palsies).

The cases resulting from birth injuries have a different history. While the paralysis is not present for some time, instead of being found at birth, if careful investigation is instituted, a difference in the motor power of the limb will be discovered; convulsions begin at once, or within a few hours, or a day or two after birth, and may continue for several days and then never recur, or they may recur at intervals for years, or may continue through life. These come from irritation of cells which have escaped actual destruction. The motor loss, when found, is the same in possible distribution, but is flaccid in the congenital form, whereas there is rigidity in the birth-palsy. In the birth-palsy, when the child attempts movements with the legs, they will be found to be weak and awkward, and almost universally the first obtrusive fault will be an adductor spasm which holds the legs together. The pathology of this spasm, which is a prominent symptom in all these palsies, is extremely doubtful, since its occurrence does not seem to depend very closely upon site or area of lesions. Gowers thinks that it depends less upon the injured parts, than upon the necessary control, which now is lacking, of the parts unaffected. It certainly follows lesions of the central ganglia, as well as of the cortex. There is the same spasm in the calf-muscles, and all conspire to make the leg rigid, and the stiffness is increased by attempts to bend it, so that the leg is moved as a whole. Another early condition in the birth-palsy is rigidity of the arms with an inversion of the thumbs, and the head may be bent back. If the rigidity is one-sided, the convulsions are also. In both varieties of palsy, walking is long delayed, if it is ever possible, on account of the adduction, and even crossing of the legs (scissors-legs.) When the child does start to walk, we find that it teeters on account of the foregoing disabilities, and the contractures which result, and also because movement of spastic muscles increases spasticity. These contractures produce in the legs talipes of some sort, usually equino-varus, but the inversion may be so great that the soles of the feet point toward one another. Spasm results in hypertrophy of the muscle involved, and so some muscles will be very weak while they are enlarged, but the muscular masses as a rule are diminished in the affected parts. When this spasm is very marked, an attempt at extension of the leg will set up an irritation of the muscles, and after a little extension the limb will jerk back. The spine may become curved by the irregularity of the muscle-supply and spasm. The knee-jerk is exaggerated, and ankle-clonus might be expected, but will be absent in those unable to walk, since the condition of extreme muscular irritability neutralizes the reflex.

Athetosis, and a finer movement which has been termed hemiplegic chorea, and tremor are also found. Athetosis consists of queer worm-like movements of parts which may begin in one part, and then go to another, and then another, or may be confined to a single part. This may be all over the paralyzed parts, and even in the face, and the platysma, but is more often in the fingers of one or both hands. These cases show an over-extension, and subluxation of the first phalanges from spasm of the interossii, and that allows the fingers a wide range of movement, which may resemble our idea of the gyrations of snakes on the head of Medusa, more than any movement of those members, with which we are familiar. This condition sometimes renders the affected person entirely helpless, as the hands and arms are unsuited to any form of labor. There is a finer range of movement which is called chorea, although that name is also applied to athetosis. Some subjects display a tremor which begins with a movement, and grows constantly coarser and wider as the movement is developed, until it renders the motion as abortive as the intention tremor of multiple sclerosis. This has been called chorea spastica, and both it and athetosis are common in the hemiplegic form of paralysis. There is no loss of sensation of the skin.

Gowers mentions seeing one case where there was a considerable defect of sight without any change in the ophthalmoscopic image. Squint is not uncommon, and seems to arise from spasm or weakness of a variable location, first affecting one, and then another muscle.

The sphincters of the bladder and rectum are not paralyzed, but may be weakened, or may be the site of spasm. The mental state being low, initially at any rate, combines with their muscular losses to make them filthy in early life, but they tend to improve as they grow older.

Swallowing is initially faulty in the congenital cases to some extent, but it is not a feature of later periods, although they drool saliva from inability at first, and from habit perhaps in later years. There is over-action of the salivary glands in some cases and the habit, which is well-nigh universal, of continually thrusting their hands into their mouths, sometimes for hours together, all contribute to the same result.

Speech is not normal in any but the very mildest cases, and the loss ranges all the way from a slight defect, which is almost completely obliterated as time goes on, to a creature who can only signify his wants by gibberish totally unintelligible to any but his constant attendants. The amount of aphasia is closely related to the extent of the mental loss.

Mental loss can be said to rulably follow the extent of the paralysis. A diplegic is prone to idiocy, while a paraplegic may be precociously bright, but it is safe to say that they are never quite normal mentally. Hemiplegics differ among themselves, showing all grades of mental equipment. It may be broadly stated that severe physical loss presages great mental deficiency, but the converse is not equally true.

Epilepsy results from secondary degeneration; it is not so common in these as in the infantile cases, but its advent indicates that at length the mind will fail clear to dementia or insanity.

Electrical reactions are normal, and the atrophies are only slight, but on the contrary pseudo-hypertrophy may ensue in the affected muscles.

INFANTILE CEREBRAL PALSY.

DEFINITION. This is a paralysis of cerebral origin occurring in the first six years of life. Gowers says that it is equally frequent in the first two years, three-fifths of all cases occurring then, while three-fourths occur in the first three years, and seven-eighths in the first five years. It is always a hemiplegia, and the cases which affect both sides of the body will be found to be no exception to this rule, but are simultaneous lesions upon the hemispheres. This variety of paralysis is separated from those occurring after the age of seven by no fanciful distinction, but upon the real basis that this is a definite stage in the process of developement of a child. Cerebral injury at this time produces a halt in the developement of an affected limb, which is not true of a similar accident at a later period, and since, up to the age of seven, the speech center is not so firmly localized on a single side of the cerebrum, permanent aphasia is almost unknown if the lesion is single, whether it be upon the left or the right side of the brain.

DIFFERENTIAL DIAGNOSIS. The differential diagnosis of the Infantile form is from Paralysis from Caries, Anterior Poliomyelitis Acuta, Cerebral Palsy from Tumor, Spinal Sclerosis (congenital), Myopathy.

SEX. Female children are more prone to this condition than males.

ETIOLOGY. Infantile Paralysis may arise from any of the causes competent to produce such a result in adult life, like massive hemorrhage, embolism, thrombosis, and diseased conditions of the vascular system. Some cases are the result of an Acute Diffuse Hemorrhagic Encephalitis, in which the hemorrhagic foci are distributed over the greater part of the cerebrum, or as the final result of an encephalitis of intrauterine or natal origin. Since the symptomatology is not assumed to differ, it seems to sufficiently satisfy the conditions if we say that such paralyses are apt to occur during convalescence, or a short time after it, in a patient who has suffered from some one of the infectious diseases, of which scarlet fever and measles are the most potent. Cases have been noted after diphtheria, pertussis, bronchitis, gastric fever, and mumps. It has come on in a day or two after a child has suffered from a fall. A majority of the cases (5 out of 8, Gowers) have been preceded by no apparent infection, and constitute the so-called primary cases which differ in no manner from the others, either in method of onset, progress, or subsequent results, and therefore rest upon a similar infective basis, although its point and manner of entrance is not susceptible of demonstration.

PATHOLOGY. Our actual knowledge is slight, since, as has been said, few die in the early stages. The lesions found have been a superficial sclerosis of the hemispheres, microgyria, and porencephaly. The usual effect of an infective agent, is to produce thrombosis and emboli, and that is the theory held by Gowers as to the process in both the primary and secondary forms, and of these he favors thrombosis as being the most probable. The occurrence of microgyria or porencephaly would rest then upon the occurrence of a vascular obstruction in a vessel of the surface, or in the mass of the hemisphere. Struempel considered that the cause of the primary cases was an acute polioencephalitis. Both Gowers and Sachs point out that the scanty post-mortem findings have not disclosed any such condition; that pathology teaches us that a primary inflammation of the cerebral nervous tissue is far less common than of the spinal, and that analogy from spinal cord diseases is not a good guide, since the spinal nervous system is not susceptible to secondary disease, while the cerebral is eminently so.

SYMPTOMATOLOGY. Primary Effects. The illness begins with or without fever, but in 50% there is a severe convulsion, or a series of them, which may be one-sided at first, and afterward general. They may persist for days and hours, or may cease for a time, and again recur. Soon after this the child is found to be paralyzed upon one side; if on both it is a double lesion. This paralysis will be found to be on the side on which the convulsions manifested themselves, or where they began, and were most severe. Vomiting may be present. This paralysis is a permanent one, but its area may be quickly reduced. After a few months it has become very much reduced, and the face and arm are apt to be comparatively free, on account of the power of compensation from the hemisphere opposite to the site of attack. In quite a proportion of the cases, however, some traces remain in the arm, but the shoulder and elbow are less liable to be disabled than is the hand. If the case is secondary to some infectious disease with much prostration, the patient may be so weakened by the shock that the paralysis is not discovered for some days. Unilateral lesions have not quite the localizing value that they have in adults, since the lesion may lie in the central ganglia. The leg always regains some power, although under the most favorable conditions there will be some limp, on account of the arrest of a normal amount of developement. The face-palsy is generally trifling, and the only trace may be an overaction of the paralyzed side. This is best disclosed by some response to the emotions where the movement will begin first on that side, while a strong volitional movement will not disclose any difference. Paralysis of a lasting nature never results, unless the motor cortex or tracts are directly injured.

If speech has been developed prior to the seizure its loss will only be temporary, and will be regained in a week or two, or at most in a couple of months. Sensation is not affected.

Knee-jerk is exaggerated, and ankle-clonus is frequent (contrary to the rule in birth-palsies), but rigidity is not so much a feature as in adult cases.

Mental changes may be as great as in birth-palsy, and they may be present, although the degree of paralysis may be slight. We may find every variety of mental obliquity, from hysteria to idiocy, and Gowers states that the worst case of hysteria that he ever saw was in a girl who had suffered from infantile hemiplegia.

LATE EFFECTS. When the convulsions with which the attack was ushered in are accompanied by loss of consciousness, with tonic and clonic spasms, and occasionally with biting of the tongue, and relaxation of the sphincters (they may resemble petit mal), the resulting paralysis is usually slight. In such cases a usual result is automatism, which is the performance of acts without apparent volition, and often is important in a medico-legal way.

In two-thirds of the cases convulsions recur at a later period of life, years after the origin of the paralysis. They most commonly occur at puberty, and forever afterwards recur at stated intervals, or dropping from sight for a few years, again recur as a fixed condition in the late "teens," when the strains of life begin to test the powers of resistance. They usually begin as a focal (Jacksonian) epilepsy, affecting only the paralyzed limb, or that side of the body, but almost certainly develope into a typical generalized epilepsy. Spratling says: "The old lesions of an infantile cerebral hemiplegia are the most frequent findings in the brains of epileptics." The percentage of diplegics, and hemiplegics who afterwards become epileptics was 44.3% (Sachs and Peterson); 26% (Osler); Wallenberg found 66 in 160 cases, while Lovett reported 26 severe cases, of which 96% became epileptic. Tucker said that he was inclined to the belief that there was cerebral cell lesion in every case of epilepsy, and that there was no such thing as an idiopathic type. He thought that most cases were due to birth accidents.

Contractures come on as power returns in a limb which has been paralyzed, and spasm is found in various muscles. There is a continued, but perhaps slight spasm in the feet, which can always be overcome by gentle pressure, but which is sufficient to over-extend the toes, and cause the person to be uncertain in gait (titubation), even if he have the requisite muscular power. It produces some grade of talipes varus, or equinovarus. The same condition is seen in the middle joints of the fingers, producing over-extension, and even a subluxation of the fingers. There is a fixed spasm at times in the elbow, and the wrist in extension.

Athetosis is seen in from 10 to 50%, and if an adult is found with this symptom it is almost certain that the paralysis from which he is suffering is of infantile origin. Not exactly the same, but similar, are what are known as associated movements. These are such movements as are repeated on the well side, when a strong effort is made on the paralyzed one.

It may be generally stated that voluntary movements are disordered and ataxic.

Atrophy, accurately speaking, is not a symptom of this disease, but there is an arrest of developement which gives that appearance to a limb. The presence of an undeveloped scapula and arm are diagnostic of infantile paralysis. The muscles are usually reduced in size in the affected area, but an exception is found in those which are the seat of spasm, since its continuance usually produces hypertrophy of the affected muscle.

PROGNOSIS. The danger to life is not very great, if we except the cases which succumb to the severity of the initial convulsions, or less often to the strain of the status epilepticus in later years. Mills differs in saying that most diplegics die in infancy or childhood, while Oppenheim says that they live to an old age. All tend to show some physical improvement, but it differs in the different types, although some cases of even the worst type will almost completely recover. The greater the defect, the later does improvement take place.

Diplegics have the worst prognosis, since the symptoms which make us take a pessimistic view of any case are most frequent, and most marked in this variety. These symptoms are convulsions, contractures, mental enfeeblement, athetosis, and epilepsy. The history of the first year gives us grounds for predicting the future, and yet some of these cases begin to develope mentally after puberty. It is not possible to give a definite prognosis about the power of walking until after the sixth or seventh year, since some children will finally walk fairly well, even if at first the progression is cross-legged. If the paralysis comes on after birth, i. e., is typical infantile palsy, the probability of mental impairment is greater, the younger the age at the time of the attack.

Aphasia has a good prognosis where it is not accompanied by mental defect, since in early years the speech-center is not so definitely and exclusively localized in the left hemisphere. Dysarthria may remain. This may be helped by training.

Epilepsy is not liable to occur, if it does not occur in the first two or three years but exceptionally it has been known to delay its advent until the tenth year. Its appearance presages eventual idiocy, and at any rate great mental impairment. Its attacks seem to grow less in mature years, and it is said to disappear about the fifth decade.

Athetosis and choreiform movements are the cause of great disability, inasmuch as they may render the hands and arms utterly useless. The appearance of these and the contractures render the outlook very gloomy, since they do not tend to pass away when once established. Bilateral chorea has an especially bad prognosis. Mere retardation of mental powers and speech should not in itself be a ground for a bad prognosis, since recovery of these may come later, but mental impairment, if continued, carries with it a probability of physical defects.

Convulsions in a birth-palsy are a ground for prognosis, depending upon their severity and persistence, since they show the depth and area of the brain injury.

In general we may say that the prognosis is good, if, after a few months, spasticity is still absent in the limbs, and there are some attempts at voluntary movement.

Paraplegic cases have a better outlook, since speech-defect is usually only associated with implication of the arms, and these cases also do not have much liability to mental losses. While the legs may be stiff and uncontrollable, patient treatment will often improve them greatly, even if walking is delayed until the sixth or seventh year. These cases are subjects for hopes of betterment, however, rather than of perfect recovery.

In all cases, the presence of the stigmata of degeneracy causes one to make a less favorable prognosis, and a microcephalic head debars us from giving much promise of further developement.

Diagnosis. The presence of a paralysis, either diplegic, paraplegic or hemiplegic in localization, and with exaggerated deep reflexes, proves that there is some injury to the motor tracts above the motor cells of the anterior horns of the cord. This makes it an upper neuron paralysis. The implication of speech, or the mental faculties assures us, among other symptoms pointing to the same localization, that the cells of the motor cortex of the brain are primarily affected. We are sure then that the case is one of cerebral paralysis. If the history was faulty or absent, the deficient developement of the affected parts, the presence of athetosis and associated movements, deficient mental developement, and recurring convulsions, or some form of epilepsy, would be strongly indicative of an infantile origin. On mere localization of the paralysis we should have grounds for assuming that a diplegic indicated a congenital case, that paraplegia was a birth-palsy, and that hemiplegia was infantile. Idiocy, as well as cranial assymmetry, is indicative of prenatal origin, while epilepsy and recurring convulsions are more common in the infantile cases, and a paralysis confined to the lower limbs (paraplegia) with an unimpaired intellect is often found in cases of birth-palsy. If we have a history, we should conclude that a paralysis was prenatal in origin, if after a normal birth the child was deeply cyanosed, or without previous convulsions, or feverish attack, should display later an extensive paralysis, particularly if it was a diplegia; that it was a birth-palsy, if an infant, after a difficult, precipitate, or instrumental birth was at once, or very soon, seized with convulsions which were long-lasting or recurrent, after which a paralysis was discovered; that it was infantile, i. e., before the seventh year, if after a history of good health the child became the subject of a hemiplegia, without warning, or during convalescence from an infectious disease, even if some time had elapsed from apparent recovery, and if the aphasia accompanying it was very transient.

DIFFERENTIAL DIAGNOSIS. Spastic Paralysis from Caries of the Vertebræ may closely resemble cerebral spastic paralysis, but in this case the sufferer from caries had a history of normal physical power, and an examination of the spine will reveal a tender area where motion is restricted, and there will usually be disturbance of temperature and localized pain. A skiagram will reveal pathological changes in the spine.

Anterior Poliomyelitis. Some cases classed as cerebral palsies of children are actually unusually extensive cases of anterior poliomyelitis. The infantile type is often an instance of this disease in its widespread form, and no certain distinction can be made: but from the congenital and birth forms differences will be found in the fact that the child has had a period of normal life. Poliomyelitis will generally, although not always, present a history of a febrile attack preceding the palsy. There is also marked wasting of distinct muscles, while others remain plump and well-nourished, and it is a flaccid, and not a spastic paralysis; there is loss of deep reflexes, and of the reaction of the muscles to faradism. A whole limb is not so evenly affected.

Spastic Paralysis from Tumor. When the history reveals that the paralysis has been gradual in onset, and without symptoms of an acute antecedent disturbance, the presence of a tumor should be regarded as a possibility.

Spinal Sclerosis. (*Congenital.*) In some cases the damage to the nervous system of an infant in utero is expended upon the spinal, instead of the cerebral structures. Generally this takes the form of congenital syringomyelia, and is not likely to be confused with the diseases under discussion, but two cases are noted by Dejerine which show that such a localization may be a source of error. The first case was diagnosed as a congenital spastic quadriplegia, lived until the age of 63, was intelligent, and was supposed to be suffering from the effects of a double hemispherical lesion. On autopsy was found to have a spinal syphilitic lesion only, the brain being normal. His second case also proved to have a normal encephalon. The diagnosis rests, in a spinal localization, on integrity of the intelligence, absence of paresis and spasm of the face, and absence of convulsions.

These may also be the conditions in a typical Little's Disease, if the arms are not involved. If they are, we shall have involvement of the face, strabismus frequent, intelligence affected, and epilepsy frequent.

Myopathy may look like it, but it is not hemiplegic, the knee-jerks are not exaggerated, there is no history of an acute attack, nor have there been any convulsions.

TREATMENT. This is divided into several groups on account of the difference in method of onset, and also from the fact that there is no relation between the measures necessary to antagonize the early and the late features of the condition. When the palsy is the result of some pre-natal condition which has run its acute course before the birth of the child, our treatment will be that of antagonizing results. If it is a case of birth-palsy, the infant will suffer from convulsions at its birth. These are recurrent, and very obdurate; the mortality is extremely high, and there is little scope for symptomatological prescriptions. If a few doses of Belladonna, Arsenicum, Cuprum ars., or Hyoscyamus as indicated do not relieve them, more drastic measures should be employed. Compression of the carotids against the hyoid bone will reduce the flow of the blood to the brain: the pressure should be continuous, but not persisted in for more than an hour. Chloroform and Amyl nitrite have been used, but not with very favorable results. Cold applications to the skull are not of much use. Chloral hydrate, 2 grains, and Potassium bromide, 5 grains, were given every half-hour to two hours to a case of my own, with good results. The hot-bath is not indicated in this condition, but in cases of doubtful etiology it can do no injury. When the case is infantile, and at a later period of life, the treatment should be that of an apoplexy in an adult. In these, as well as birth-cases, great care should be exercised that position should never be such as to impede the venous return from the brain. If there are good grounds for the localization of the clot, the skull should be opened, and the clot washed out. It is advisable to take long chances in this procedure, since the hemorrhage is meningeal, and the after history of these patients, if they recover from the initial shock without operation, is one of harassing invalidism, since they surely will have some grade of spastic paralysis, and generally will also be epileptic. The treatment of the late stages of these cases is the same for all, as it comprises all the possibilities when we conserve the general nutrition of the body, the special nutrition of the limbs, combat deformities, and mitigate as much as possible athetosis, spasms, and epilepsy. The general care should involve the most assiduous training of mind and body, and sometimes results more happily than we should expect, from ingenious substitution of normal parts in the offices of the impaired ones. In this department, as well as in the more specialized ones, it will often be found that the constant care of a mother will accomplish more than the occasional treatments of a more skilled operator. The nutrition of the limbs may be very greatly improved by massage several times a day, immersing the limbs in hot water for ten minutes before the massage, and by faradism to the extensors in the upper limbs, and to all the muscles of the lower. The feet should receive great attention, since abnormal positions of the toes and feet are often a great hindrance to walking. Phosphorus 6x, is a remedy which may be of use in this connection. Much can be done by these means, *if* education is co-incident, and painstaking. All this must be begun at an early date to be of much value. Contractures should be antagonized by splints, and tenotomies, and lengthening of the tendons, and this must be early in the birth-cases, since they do not respond as well as do the infantile. Tendon-transplantation has not been of as much value as in the contractures of the adult, but the substitution of normal muscles for impaired ones by tendon transplantation results more happily than one would expect. Section of the spinal roots in the cauda equina has been practiced for the relief of spasticity, resection of nerve to definite muscles, and the re-education of the antagonists. isolation and injection with alcohol of the nerves to the spastic muscles. The cortical centers have been excised when they could be accurately located, and with the substitution of a flaccid for a spastic paralysis. Nerve-stretching, and plaster of Paris splints are useful in the treatment of athetosis. Generally, however, it is irremediable. In the use of electricity, the applications should be made with no current until the child is free from terror at the procedure, and then only such currents should be used as do not induce painful shock. The efficiency of remedies is very doubtful, but some clinical reports lend color to the belief that they have mitigated conditions at times.

THERAPEUTICS. Baryta carb. This is a remedy for deficiencies in nervous tissues, and has a symptom tension in the calves, and muscles of the back.

Causticum. Contraction of flexor tendons. Contractions of the tendons of the fingers and twitchings in them, and also in the tendo Achillis, with a marbled appearance of the skin. Contraction of the plantar muscles. It certainly does favorably affect the nutrition of nerve-cells.

Calcarea carb. The symptomatology indicates that this may be useful in the birth-cases, and the ante-natal ones, where there is a faulty developement of the motor tracts.

Plumbum. This has been recommended, but it has a flaccid, and not a spastic form of paralysis.

Secale. This remedy is indicated for a spastic form of paralysis, with a cold surface and hyperesthesia, so that touching the skin arouses twitchings and jerkings. There is a spasmodic curvature of the arms, with a distortion and turning back of the fingers, and the same in the legs. A convulsive spreading-apart of the fingers, with the other symptoms, might point to its use in athetosis.

Silicea. It is a specific for the re-absorption of scar-tissue in nervous structures.

Sulphur. This is on constitutional indications, and is not to be ignored.

Lathyrus. This produces a general spastic condition.

Graphites may prove efficient as an absorbent.

The late epilepsies should be treated as is indicated under that caption.

CHAPTER XI.

THE APOPLEXIES.

CEREBRAL APOPLEXY FROM HEMORRHAGE.

DEFINITION. It is that combination of symptoms which results from the rupture of an artery in the cerebral meninges, or in the substance of the brain, or brain-stem. It results in a motor or sensory palsy (or combination of the two), of greater or less extent, and secondary motor changes resulting from subsequent degenerations; mental changes probably.

THE SYMPTOMS OF A TYPICAL CASE. There is sudden unconsciousness lasting for two or three days, paralysis of the whole body, and a suspension of all the functions, except those of the heart and lungs. The pulse is strong, full, and slow, and the respiration is stertorous. In two or three weeks the general paralysis has become a hemiplegia, and the patient later regains a moderate degree of health with weakness and contracture of the paralyzed side. He dies after two or three repetitions of the attack.

DIFFERENTIAL DIAGNOSIS from Syncope, Toxemias, Poisons, Alcoholic Intoxications, Uremic Coma, Epilepsy, idiopathic and focal, Hysteria, General Paresis, Diabetes, Acute Softening from Thrombosis and Embolism, Fracture of the Skull.

AGE. While it is possible at any time of life, it is rare before the fortieth year, and is most frequent in old age.

SEX. Males more often than females.

ETIOLOGY. This varies somewhat according to the site, since we must recognize that hemorrhage may take place from rupture of the arteries and veins of the membranes, as well as from the central arteries of the brain, cerebellum and brain-stem. If the cranium sufferes severe injury, it is apt to be near the vertex, and may cause a rupture of the middle meningeal artery, or one of the veins in its course to the sinus. The hemorrhage will generally be epidural, i. e., lying between the cranium and the dura, and no previous disease of the vessels is necessary for its occurrence. Such violence may also cause a hemorrhage into the arachnoid space (intradural, or subdural); such a hemorrhage is far more frequently seen in the alcoholic or the insane. Similar hemorrhages occur in the pia, and like the intradural indicate that the vessels have previously been the seat of degeneration from alcohol, syphilis, some of the infections, or the arteriosclerotic changes incident upon old age. Traumatism may also be a cause. Under such conditions the weakened vessels may give away from an emotional strain, some extra muscular exertion, straining at stool even, or be precipitated by some slight injury. The great bulk of hemorrhages, however, occur in the central arteries of the brain, affecting the corpus striatum, thalamus, and internal capsule. Charcot said that next to these in frequency are hemorrhages in the narrow band of gray matter in the external capsule, called the claustrum. The sites next to these in frequency are in the pons, medulla and cerebellum. In all this large group an antecedent weakening in the arterial coats is a necessity, and the rupture is in many cases spontaneous, or it may be due to the strain from a hypertrophied heart, the strain imposed by the impaction of an embolus, or the vicissitudes of ordinary life. In the senile, while we may find arteries which have simply thinned out without evidence of definite disease, more probably we shall find atheromatous change, where fatty degeneration has weakened the artery, or it has broken down from the completeness of the process, or calcareous changes have made it brittle. This form of disease does not affect the smaller arteries, but they are weakened in two different ways. In the first place they may be subject to a degenerative arteritis, which results in the formation of multiple aneurysm, hundreds sometimes, any one of which is the site of possible hemorrhage. This is very common in the central arteries, and in the degenerative period of life. The small central arteries are also affected by fatty degeneration from infections, exhausting diseases, purpura, marasmus, and this is the cause of apoplectic seizures in infancy, and in young persons suffering from such conditions.

PATHOLOGY. At the site of all hemorrhages, except the meningeal, which are often traumatic, we shall find evidence of some sort of arterial change. After the hemorrhage occurs, there will be a difference in pathology, from the variation in site. If it is extradural, the blood is generally retained, by the resistance of the tissues, in a more or less small globular mass, which compresses the underlying brain; it tends to become absorbed, or to form a cyst. If it is intradural or subdural, it is more diffused, but still circumscribed in area exerts localized pressure upon the brain, and generally sets up a reactive inflammation in the meninges, eventuating in a matting together of the structures in the neighborhood, affecting to some extent the circulation, and generally terminating in the formation of a cyst. If the hemorrhage is cortical, it is rulably small, and the extravasated blood spreads out in a thin layer, which may be circumscribed, multiple, or may sometimes envelope the whole hemisphere. It may remain fluid and unchanged for weeks. A sub cortical or central hemorrhage tears out a portion of the soft tissue of the brain, from the bulk of a hazel-nut, up to the size of one's fist.

If the hemorrhage is in the neighborhood of the central ganglia, it is liable to break through into the ventricles, where it may consolidate into a perfect case of the cavity, or forcing its way downward rush through the iter, and tear out the soft tissues of the fourth ventricle. Pontile hemorrhages generally occur in the middle line. Cerebellar hemorrhages are principally the result of the rupture of the superior cerebellar artery, and usually progress externally, and break into the fourth ventricle. The intrusion of this mass of blood into the brain is followed by an edema of the surrounding areas, which become the site of numerous capillary hemorrhages, on account of the change of blood pressure, and the local inability to resist it. The clot begins to lose its bloody color in from two to five weeks, but remains hard for about four weeks. In all cases by the fifth week it is yellow, as a result of the activity of the leucocytes. These cells, enclosing blood-corpuscles become evident about the third day. From the fifth to the twelfth week it is a yellowish mass, and then becomes ocherish in color, from the presence of free pigment. While after a small hemorrhage a cyst-wall may appear in a month, and though contraction may begin a little later, it may be from one to two years before contraction is accomplished, and the spot is quiescent. The cyst-wall is formed from glia and connective-tissue. This cyst may undergo fatty degeneration, and replacement by fibrous tissue, or it may remain a cyst, filled with straw-colored fluid. Obliteration is rare after hemorrhages of much size. This area of changing tissue is essentially a foreign body, and, by its pressure may more or less completely inhibit the function of the surrounding portions of the brain. The fibres descending from injured motor cells begin to degenerate at the 10th to 14th day, and in old cases the degeneration has been found to extend down the cord. As in all other lesions of nervous tissue, the replacement is by structures incapable of nervous function.

PHYSIOLOGY. The initial state of unconsciousness, shock, and universal paralysis which mark the early days of an apoplectic attack is the result of the pressure of the clot (Niemeyer), plus the disturbance of the general cerebral circulation (a prominent factor—Gowers). However, pressure can actually produce the effect. Tests have shown that a pressure of 130 mm. on the surface of the brain of a dog will render the animal unconscious, while one-tenth of that pressure will produce the same effect if exerted suddenly. Slow exudations are compensated for by a relative decrease in the cerebro-spinal fluid. The pressure of blood in the arteries is about 150 mm. of mercury while the density of the brain tissue is about 10 mm. (Dana). In other parts of the body the arteries derive support from the surrounding structures, but if the arteries of the brain are not competent in themselves they will rupture if conditions are unfavorable.

SYMPTOMATOLOGY. Premonitory symptoms are rare, except in pontile or cerebellar hemorrhages, not being like thrombosis, where we may see for a period of hours or days some slight dizziness, fullness in the head, bad dreams at night, headache with epistaxis, disturbance of heart's action, and a little numbness of the hand or foot upon one side. Occasionally we do see definite symptoms and they then arise from an exaggerated degree of arterio-sclerosis, and differ from those just given in the fact that they are of more gradual onset, while those of thrombosis are present for a much more limited period. The only source of error would arise in cases like that quoted by Hardy, where there was a headache for ten days, and then a hemiplegia. The autopsy showed that there had been a hemorrhage in two stages, both of them occurring without loss of consciousness. The rule is, however, that before the attack the patient is often remembered to have said that he was feeling unusually well, probably because the heightened blood pressure more adequately nourishes the brain than has been the rule. The attack is pre-eminently a disturbance of consciousness, perhaps slight, followed by hemiplegia. Pain in the muscles of a whole side have preceded an apoplexy for from twenty-four to forty-eight hours. In a typical case it is generally the fact, and typifies the popular conception of the word "stroke," that the victim drops in unconsciousness, with unequally dilated pupils, which do not respond to light. The deep reflexes are exaggerated for a few moments, and then obliterated, together with the superficial ones. The heart is tumultuous for a few moments, but after a little time the pulse is tense, full and slow, the face congested and bloated. The temperature is normal, or subnormal. Urination and defecation may occur, and swallowing is impossible. Isolated cases may occur, where the patient runs a few steps in an aimless manner, grasping the head, and cries out as if conscious of an injury to the brain, but this is more common in embolism. At times a patient may grow confused, complain of palpitation, numbness in a part, loss of sight and hearing, become thick in his speech, have relaxation of the sphincters, and then develope complete coma; a convulsion may be the initial symptom, usually indicating a meningeal or cortical hemorrhage, but some or all of these symptoms will be present in every case. Breathing is stertorous and slow, and will become Cheyne-Stokes in rhythm if a fatal issue is impending. If this embarrassed respiration persists for more than three days, mucus will collect in the lungs and trachea, and death will follow from the results of this; i. e., inspiration pneumonia. After the first brief tendency for the eyes and head to turn to the paralyzed side, there is a disposition to fix the eyes and the head (conjoined deviation has a common center in the cortex) toward the lesion, and away from the side which has suffered the paralysis, but the contrary is generally the fact if the lesion is in the pons. In analyzing this symptom we must be careful to distinguish it from the spasmodic twitching which may be present, which is an irritative symptom. This conjoined deviation is a temporary symptom, passing away as consciousness returns, in a day or two generally, and is due to the unopposed action of the unparalyzed muscles. For a day or two the urine is increased, and for the first twelve to twenty-four hours usually contains sugar or albumin. According to the severity and extent of the hemorrhage, this group of symptoms will endure from an hour or two to three or four days. During this period it is often difficult to determine which side of the brain is the seat of the hemorrhage, since at this time the whole body is the seat of a general flaccid paralysis, but by the exercise of care, it may be discovered that one side is more limp than the other, and that if that member be lifted from the bed it will fall more heavily than the other. Where diagnosis of the side paralyzed is difficult, examination of the abdominal, corneal, and cremasteric reflexes will disclose their absence on the side paralyzed. Another test is by placing the limbs in an uncomfortable position. After a time they will be adjusted more comfortably upon the well side, but will lie unmoved on the paralyzed one; Marie-Foix sign (see page 483). After this initial period of shock, the coma will lessen, and the well side will begin to show evidences of power. Deep coma is replaced after some hours by the lighter grade of unconsciousness which we term somnolence, and in this state the corneal, and other reflexes become evident again, the patient can swallow, and execute some movements of a slight nature on the well side. If consciousness does not return in two or three days, the shock to the brain has been so great that the nutrition is sure to suffer, joints may be affected and bedsores will also occur; and that is also true when there is hemianesthesia. The pulse and temperature will now begin to rise. The initial fall of temperature was from a little below normal down to 97, or 96 even, and the subsequent rise is apt to begin in from three to twenty-four hours. It soon attains a height of 103 to 104 degrees; if it does not stop at this point, but goes on toward, or up to 107 or 108, it becomes a sign of a complicating pneumonia, and a continuance of the subnormal state points just as certainly to a fatal issue. The skin now becomes moist, and headache begins to be evident; there is some delirium. There are all grades of this condition, but by as much as the lesion is removed from the cortex, by just so much is this disturbance of consciousness lessened, and the resemblance to a spinal lesion increased. Vomiting has been a symptom, and is more apt to occur as the lesion approaches the cerebellum, or bulb, or invades the ventricles. The paralysis will vary in extent and severity according to the location and amount of the causative hemorrhage, but it will always be of one side of the body only, being some grade of hemiplegia of the face, arm, and leg type; and even when the whole body is paralyzed by a double cerebral lesion or ventricular hemorrhage, in either case the result will be a double instead of a single hemiplegia. Speech is notably affected if the paralysis is of the right side, since it will arise from a lesion of the left hemisphere, which is the side upon which speech is developed, unless the patient is left-handed. It is either a dysarthria from paralysis of the lips, tongue and cheeks, or of the finer muscles of articulation, or an aphonia, or it is an aphasia from destruction of specific centers, or of the association tracts. Specific localizations for varieties of aphasia have recently been questioned. Hemorrhage in the central ganglia of the left side, or in the internal capsule of the left side, will cause it, and the more extensive it is, and the nearer the approach to speech centers (perhaps interference with the lenticular nucleus alone), by so much will the aphasia be marked and permanent. Exceptionally a paralysis may always remain flaccid, but usually a rigidity ensues sooner or later. Rigidity is of two types: viz., early and late. Early rigidity may come on temporarily in the reaction stage, and is a sign of irritation from secondary inflammation, rather than of destruction of the motor cells. Swallowing is impossible in the stage of coma.

When the reaction which has just been described is fully accomplished, symptoms of a more permanent character begin to be evident. The condition is now a hemiplegia of some definiteness. On examining the face one will notice that the expression is changed. The lines are smoothed out, and the eye is more open on the paralyzed side, the corner of the mouth droops, and the saliva is not swallowed, but dribbles from this side of the mouth. Raise a member on this side of the body and it falls limp, showing the absence of power in the muscles, but they may still be the seat of irritation displayed by twitching of the limb. The superficial reflexes are still absent, but by the second day the deep reflexes will show signs of the exaggeration which will be present permanently, or at least for a long time. It is possible that there will be a "crossed knee-jerk," i. e., on tapping the patella of the paralyzed side the other leg will be jerked forward. Ankle-clonus becomes evident, and after the first few hours there is Babinski's reflex, i. e., on stroking the sole of the foot there is extension of the great toe, and less often of all of them. This will be permanent. On account of the fact that the members upon one side of the body are represented to some degree on both sides of the brain there will be a weakness of the well side of the body, and the reflexes will be to some extent exaggerated. The paralyzed side will at first show diminution in the appreciation of touch and the other sensations up to a complete anesthesia, but unless the lesion is in the posterior third of the internal capsule (which is the sensory division) of the parietal cortex, or be an occlusion of the posterior inferior cerebellar artery, it will pass away as consciousness returns. Hemianopsia (blindness of one or other half of both eyes) is at first more commonly present than is supposed, but it passes off when consciousness returns, unless the visual path is actually involved in the lesion. When hemorrhage involves the path of the fibres of any special function or sense, such as hearing, taste, or smell, we shall find a like loss of that sense. If a lesion of the motor area is incomplete, and therefore irritative, we shall find tremor of a rhythmical or intention type (like that of multiple sclerosis), ataxia, athetosis, or choreiform movements. In such cases the lesion is certainly in the upper motor neuron, and probably in the basal ganglia, especially often in the optic thalamus, or is such that it impinges upon the motor fibres in the internal capsule, which is held by some to be the actual cause of all athetoid movements.

After a few weeks the paralyzed limbs begin to be capable of some useful amount of motion, more on the flexor than on the extensor side, and the legs gain more rapidly than the arms on account of their more complete double representation in the brain, and in all grades of recovery the legs retain this preponderance. The leg can generally be drawn up by the second or third week, and in cases of a slight degree of severity, the patient can walk in from a month to a year. The face also gains more than the arm, for the same reason that it also has a double representation in the brain. It may regain its power of expression in two months. At the outset the entire half of the face may be paralyzed, but except in the rarest cases, the lower half of the face only is involved after the first day or two. The forehead can be wrinkled and the eye closed, as well upon the paralyzed side as upon the other, or with only a slight degree of incapacity, but while attempts at voluntary motion increase the deformity, it will react normally to the play of the emotions. The only exception to this rule is when there is a definite lesion of the emotional motor centers (the basal ganglia, particularly the optic thalamus). In all these instances, however, the normal condition of the forehead and eyelids proves that it is a central loss, and that it is not due to a lesion of the 7th nerve, or of its nucleus, in which case the upper face is as immobile as the lower part. The arm, since it is represented on one side of the brain only, will therefore show the slowest and least perfect recovery, and the finer motions of the hands last of all, if they are ever restored. Motion of the eyes is not affected, after the

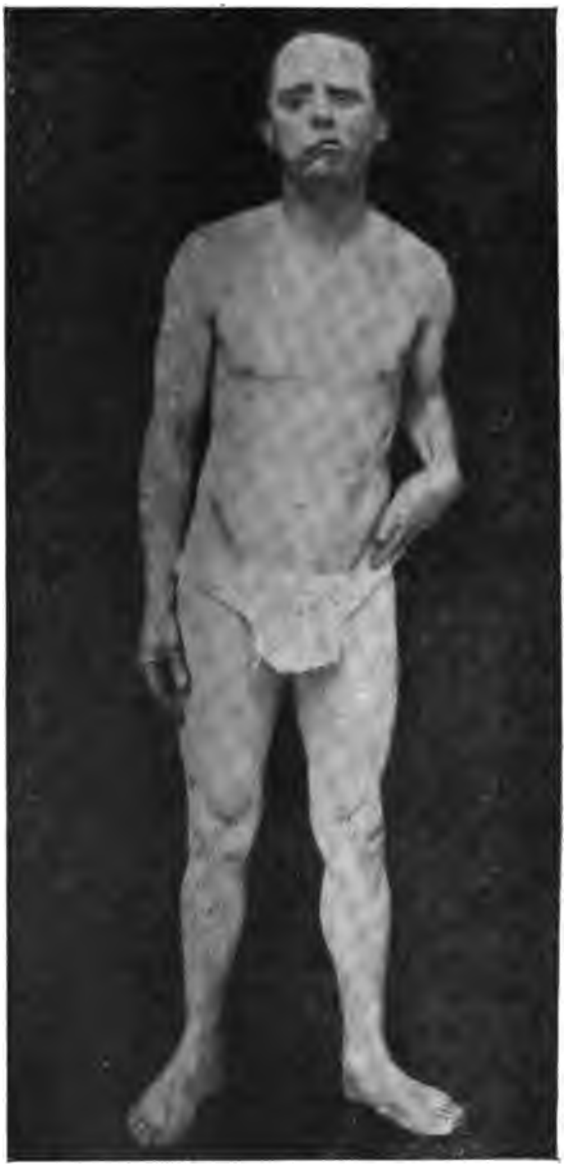


Figure 80. Secondary Contractions of Face, Arm, Hand, Foot, and Leg in Case of Chronic Left Hemiplegia. (Philadelphia Hospital.)

initial conjugate deviation previously referred to, unless the lesion involves the crus or corpora quadrigemina. Lesions in the pons will cause a defect in articulation like a motor aphasia. Secondary degeneration of the motor fibres begins in from ten to fourteen days after the apoplexy, and in one to four months we find what is termed late rigidity. This late rigidity sets up changes in the muscles which produce contractures, which are more evident in the arm than in the leg or face. Clark says that this is not an exaggeration of spasticity; but that the exact mechanism is unknown. Contractures in the leg are in extension; contrary to the rule in spinal lesions; flexion being found only in patients who are bedridden. In the upper arm there is adduction, while the lower arm is flexed, with the hand pronated and slightly flexed. The proximal phalanges are somewhat flexed, while the middle and distal ones are strongly contracted. Contracture is especially marked in cases where the hemorrhage breaks into the ventricle. When the face is contracted it is so drawn that the well side looks to be the weaker. Equal enlargement or contraction of the pupils is diagnostic of no particular condition, nor is divergence, since it may occur in normal sleep, but inequality is a sign that there has been cerebral mischief of some kind.

There is no reaction of degeneration in the muscles, nor any atrophy, except such as comes from disuse, nor is there fibrillation, since all such changes rest upon a degree of degeneration in the nuclei of cranial nerves, or the cells of the anterior horns of the cord, which in the present condition are intact, or only remotely affected, and to a slight degree, except in the rare instances where the nuclei in the pons or medulla are the site of the hemorrhage.

COURSE. An apoplexy has three critical points in time: 1st, death may occur in five minutes, and is quite likely to ensue in the attack, or within the first two or three days after it; 2d, after this the case may go on for a period of from two to four weeks, when it may take either one of two courses: (*a*) the patient may now die of exhaustion, or of some complication engendered by the paralysis; or (*b*) he may continue to improve until about the eighth week; when, 3d, it is clear that he will make a relatively complete recovery, or will remain in a state of chronic enfeeblement, which by this time has become pretty clearly outlined (Dana).

Starr sketches this as the course of an ordinary initial case. Some voluntary motion by the fourteenth to the twenty-first day. The first evidence is ability to draw up the leg, then to stretch it out, then next ability to move the ankle, and finally some flexion of the elbow and shoulder. The finer movements of the hand and foot are regained last of all, if ever.

PROGNOSIS. This is generally favorable for life in the first attack, if it is of moderate severity, but a recurrence is almost inevitable, and a third or fourth attack is rarely survived. A rapid and excessive rise in temperature, following the initial fall, is a sign of pneumonia, and an early fatality. A continued fall of temperature after the first few hours is of bad omen, as is difficulty of swallowing after several days have elapsed, or if consciousness is not regained in the first three days.

Mental symptoms are very likely to occur, and may be permanent. An ideational apraxia, inability to successfully move the limbs in purposeful movements, in a hemiplegia is an important indication of an eventual dementia.

DIAGNOSIS. In order to make a diagnosis, we must decide two things: First, is it a cerebral hemorrhage; and, secondly, what is its location? We are led to believe that it is a cerebral hemorrhage if there is a sudden paralysis of a hemiplegic type, accompanied by great mental confusion, or some grade of unconsciousness, not dependent upon the failure of the heart's aciton. If the coma is deep the discovery of the hemiplegia may be difficult, but will be revealed by the Marie-Foix reaction, viz., if the foot is pressed into extreme plantar flexion, and at the same time the toes are compressed it will produce a sharp contraction in the tensor vaginæ femoris, which will not occur on the paralyzed side. Lumbar puncture will usually reveal blood in the cerebro-spinal fluid. The retinal arteries will be found to be engorged. Blood-pressure will be found high. The only exceptions to a hemiplegic localization of the paralysis will be in those exceedingly rare instances where the lesion is entirely sensory, or in the case of a primary localization in the ventricle, in which case the resulting paralysis is generally a double form of hemiplegia which is termed a diplegia.

DIAGNOSIS OF LOCALITY. Meningeal hemorrhages may be either upon the external surface of the Dura—Epidural, on its inner surface—Subdural or Intradural, or of the Pia—Subarachnoid, Pial or Cortical. These may be either traumatic or idiopathic, the epidural being almost always traumatic, the subdural probably so, while the pial is usually idiopathic. Any severe injury to the skull is likely to rupture a meningeal artery or one of the veins of the dura on its way to the longitudinal sinus, as most of these injuries are on the vertex, but the pial vessels do not always escape. All cases of meningeal hemorrhage are apt to produce convulsions, or spasmodic movements of one half of the body, or parts of it at least. In the traumatic cases unconsciousness may not come on for hours, but it is more common to see a short period of unconsciousness, and then an interval when the person appears rational, and may go about his business for days or weeks, and then relapse into coma. The symptoms do not differ greatly from those of a central apoplexy except that the temperature is normal or a little elevated, instead of showing an initial fall. The pulse is full, and hard, and slow, and the tension is high. Breathing is stertorous, and changes to Cheyne-Stokes if the clot is large, and compression great. The reflexes are usually exaggerated, and there is considerable rigidity, because the lesions are usually irritative, and not destructive. The eyes are turned toward the lesion, the pupils are contracted, most marked in the eyes on the paralyzed side, except in case of fracture at the base which results in pressure upon the 3rd nerve. This produces what is known as the Hutchinsonian pupil, i. e., there is an inequality; the pupil is ***dilated on the side of the lesion, and contracted*** on the side which is paralyzed. Speech is affected as in ordinary central cases. Traumatic cases are usually fatal if not operated; the mortality then is not over one-third to one-quarter. If left alone the coma deepens, breathing becomes stertorous, the pulse becomes rapid and weak.

Subdural hemorrhage is far more likely to be the result of a slight injury to a person whose arteries have been weakened by alcohol, syphilis, or insanity, than to be the result of traumatism alone. The alcoholic is extremely liable to falls, or to receive a blow in an altercation, while the insane person is exposed to all sorts of injuries from his own acts, and from those restraining him. The symptoms are very variable, since the two factors of compression and irritation are in variable proportion, compression predominating greatly, and the hemorrhage may be confined within a small area, or be very diffuse. There is generally a period of headache or vertigo, and then the patient becomes comatose, hemiplegic, and sometimes displays anesthesias, rigidity, and twitchings. At other times the paralysis will be barely noticeable, but the patient will be somewhat comatose, with the muttering delirium of a patient suffering from "wet-brain," or edema of the alcoholic. In dual hemorrhages occurring in paresis, the patient becomes comatose without warning, he often has some convulsive movements, and a hemiplegia developes. In these cases there is very often rapid improvement, and the patient gets partially well, usually experiencing other attacks latter.

Pial or Cortical. This is a rare condition and usually occurs after the membranes have been previously thickened by syphilis, alcohol, or the changes due to old age. Traumatism or overstrain is usually demanded as an exciting cause. Diagnosis from the foregoing is very difficult. Stupor or semicoma may be present, or consciousness may be undisturbed. The hemorrhages are generally small, but if large may extend into the white tract-areas. Since the cortical representation of the whole of any one member covers a relatively large area, if a hemorrhage occurs over a motor area the paralysis will be a monoplegia, or an incomplete hemiplegia. These lesions are rarely destructive, but are irritative, and we therefore are apt to see localized spasms or twitchings, and if we find convulsions they will be Jacksonian in type. They will be unilateral,

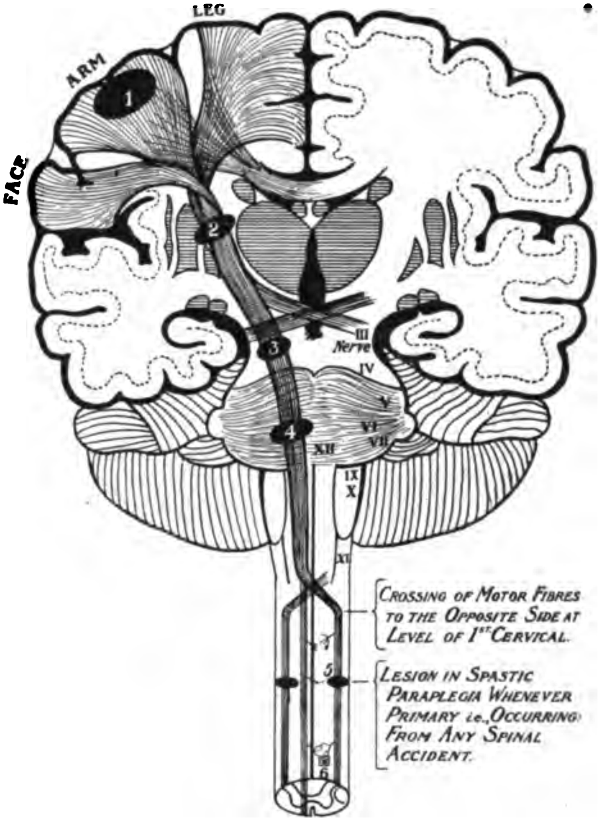

LEG
ARM
FACE
1
2
3
4
5
6
III Nerve
IV
V
VI
VII
XII
IX
X
XI
Crossing of Motor Fibres to the Opposite Side at Level of 1st Cervical.
Lesion in Spastic Paraplegia Whenever Primary i.e., Occurring From Any Spinal Accident.

FIGURE 81. LOCALIZATION OF THE DIFFERENT FORMS OF APOPLEXIES. It will also explain the Symptomatology of many Tumors.

1. Cortical or Subcortical Apoplexy; gives rise to monoplegia, or localized anesthesia, and convulsions. 2. Capsular apoplexies; give rise to hemiplegia, or hemianesthesia, or both. 3. Hemorrhage into the Crus; this gives rise to a paralysis of arm or leg, or a hemiplegia of the opposite side, and hemianesthesia, with rigidities and ataxia. It also causes a paralysis of the motor oculi on the same side as the lesion. 4. Represents a lesion of the lower third of the pons, and typically gives rise to a crossed or alternate hemiplegia; since it will almost necessarily cut off the fibres of the ascending sensory root of the fifth nerve, it will produce an anesthesia of the face on the side of the lesion, and on the opposite side of the body, since there is a sensory crossing in the lower part of the medulla. Since the lesion affects the fibres of the middle peduncle of the cerebellum, there will be rotation of the body to the side of the lesion. Any lesion between 4 and the level of the lower margin of the cerebellum, as shown in the cut, will produce this facial anesthesia, sparing the upper part of the face more and more completely as the site of the lesion becomes lower.

except in the rare cases where the hemorrhage is bilateral. There is a conjugate deviation of the eyes toward the side of the lesion, a primary rise of temperature, instead of the initial fall found in the central apoplexies, and therefore the temperature picture is like that of thrombosis, embolism, or hemorrhage into the pons and medulla.

Subcortical. A small hemorrhage just under the cortex will give rise to a group of symptoms which are partially irritative, and partially destructive, and therefore, if it occurs in the motor zone, there will be the irritation symptom of a persistent spastic hemiplegia, or hemiparesis. If in the sensory area, we shall find exaggeration, or diminution of sensibility in the areas innervated from that locality, according as the lesion is destructive or simply irritative to the sensory cells.

Centrum Semi-ovale. This is rather frequent in the senile state, and gives rise to symptoms of irritation of the sensory, motor, or association tracts.

Capsulo-ganglionic. This is the typical cerebral hemorrhage, and is marked by unconsciousness, hemiplegia, incomplete recovery, and almost certain recurrence. If the rupture has occurred at the knee of the internal capsule, or at the anterior two-thirds of the posterior limb, we shall have a motor paralysis of the opposite half of the body. If the rupture is a little posterior to this, there will be a combined motor and sensory paralysis of the opposite half of the body, while if the posterior third only of this posterior limb is affected, we shall have a paralysis of the opposite half of the body entirely sensory in character. This is very rare, but if present, we may have an ataxic form of hemiplegia without any actual motor paralysis.

Thalamus. Hemorrhage in this ganglion will generally exert sufficient pressure upon the contiguous motor tracts in the internal capsule to produce a temporary hemiplegia, but the deep reflexes are not usually exaggerated. The peculiar symptoms relate to the sensations and emotions, and automatism. There will be anesthesia, perhaps dysesthesia (a dullness of sensation, or painfulness of a sensation not usually painful), which tends to be lasting, and pains in the extremities. Sometimes there are choreiform or athetoid movements, but these are probably due to irritation of the motor tracts. Hemianopsia may also be present. In ordinary hemiplegia the paralyzed face will respond to emotions, as laughing or crying, but this emotional response is lost if the basal ganglia, the thalamus especially, is involved.

Ventricular. The primary form is rare, while the secondary is much more common. Primary is caused by attempts at hanging, or by congestion from other causes which equally block the emissary veins. It may arise from concussion, and not be evident for days after the injury, or it may be the result of purpura, or pernicious anemia. The rupture may be of the choroid-plexus, velum-interpositum, or a vein of the wall of the ventricle, and from such causes as would naturally be expected to provoke one of the more common types of cerebral apoplexy. It may occur in the young, even in infants at birth. The symptoms are stupor, sluggishness, paraplegia, or quadriplegia, and a liability to general spasms without any preceding stage of unconsciousness; spasms following unconsciousness would lead one to suspect a hemorrhage in or on the cortex. A small hemorrhage confining itself to one ventricle may cause very light symptoms, but if it invades all the ventricles, as is the usual rule, we shall find not unconsciousness, but dullness, paraplegia or quadriplegia, accompanied by rigidity, and at times by spasms. General convulsions do not occur in adults except when the hemorrhage is ventricular, or of both hemispheres, but they are common in children. If the force of the hemorrhage is great, or the quantity large, it will invade the cavity of the fourth ventricle, or violently push before it the fluid normally filling the ventricular cavities, and so injure the nuclei of the cranial nerves originating there that it generally causes death. An alteration of the respiration and the action of the heart is a sign of such invasion, and this is the result in one-third of the cases of cerebral hemorrhage. Secondary ventricular hemorrhage results from the extension of a hemorrhage originating in the capsule, or its neighborhood, through the yielding tissues into the cavity of the ventricle. Such cases fail to come out of the initial state of coma, but when a day or two has elapsed after the primary apoplexy, the coma deepens, the temperature rises to 103 or more, the respiration becomes Cheyne-Stokes in rhythm, the pulse grows tumultuous, and the patient dies. This has often been wrongly interpreted as Ingravescent Apoplexy, which is a condition where an apoplexy, normal in its after history, takes from a few hours to a day or two to develope.

Cerebellar. One to two per cent. of all fatal cases are cerebellar (Dana). The hemorrhage is located, in a large proportion of the cases, in the vermis; next in order of frequency, in the lateral lobes; near the corpus denticulatum; and less often in the medipeduncles. The diagnostic symptom of such a hemorrhage is a sudden shock to the general system, which may cause the patient to fall, have convulsive movements, or forced and irregular contractions of the muscles. This is usually without unconsciousness, but marked by vomiting, and intense vertigo and ataxia. He will be exceedingly weak and prostrated, much alarmed, and breaks out in cold sweats. This vertigo requires him to lie perfectly still in a horizontal position, as it is intensified by any attempt to walk, or stand, and this disability is termed astasia-abasia. On the other hand, after a few days of severe headache, he may fall in deep coma, with the signs of an ordinary apoplexy, except that the respiration is likely to be more seriously affected. If the hemorrhage is in, or near the medipeduncle, it will give rise to forced movements of the limbs, and a rigid position of them, and of the body, which will cause a flexion in either direction, which will be preserved even while lying in bed. There is immediate ataxia and weakness of the limbs, and tetanoid spasms, referred to above, will appear at times, while sensory symptoms may be absent or transient. Any hemorrhage produces a mass which in relation to the structures near it may be regarded as a tumor, and therefore, while the paralysis produced by a cerebellar hemorrhage rulably is upon the same side as the lesion, the secondary effects of pressure upon the surrounding structures may complicate the picture. If the pyramidal tracts are compressed by it, above the point of their decussation at the level of the first cervical segment, we shall have a motor paralysis of the opposite side of the body, while some of the cranial nerves may show a paralysis of the same side as the lesion.

If below the first cervical segment, there may be some hemiplegia, and if so it is on the same side as the lesion since it is produced by direct pressure upon the motor tract, but it may be wanting. In its absence the symptoms of ataxia and spasm have been ascribed to hysteria. The pressure upon the cranial nerves, and descending motor tracts may be only sufficient to irritate them, but since the tissues of the cerebellum are very soft, and the velum between it and the fourth ventricle is exceedingly delicate, it is not unusual to have a rupture into the floor of the ventricle. This will produce disturbances in the movement of the eyes, and in swallowing; also pulse and temperature changes. Death is almost certain in cerebellar hemorrhage, and is sure if it breaks into the ventricle. The vertigo in these cerebellar cases is labyrinthine in type, and can only be differentiated by the absence of previous aural disease, and by the persistence of vertigo, double vision, and a staggering gait after the prostration has passed. Nystagmus is often seen. In small hemorrhages the patient may get nearly well, but there will be staggering gait, tinnitus, and a tendency to vertigo, and tetanoid attacks.

Pontile. This is rare, and is from the causes usual in cerebral hemorrhage, except that it is not so commonly from syphilis. It is from the median basilar artery as a rule, but may arise from the transverse. In about one-half of the cases the hemorrhage is large enough to be immediately fatal, as it will break into the fourth ventricle. If it is small, the raphe may be strong enough to confine it to one side, and this would be the natural result of a hemorrhage from the transverse artery. In this case the nucleus of the fifth nerve is usually destroyed, and there is a sensory paralysis of the face upon one side, and of the limbs, to some extent upon the other. There may be a loss of sensation on the opposite side of the body as a preponderating symptom. The peculiarities of this form of apoplexy will be more readily comprehended if we recall that the pons is traversed vertically in its anterior portion by the descending pyramidal (motor) fibres to the whole body, and the sensory fibres lie deeper in the area called the tegmentum. The root fibres of the 5th, 6th, and 7th cranial nerves, i. e., those coming down from the brain, also lie in the anterior part of the pons. The decussation of the root-fibres of the 7th, or facial nerve, takes place just at the level of the apparent nucleus of origin of the 5th nerve. If then a hemorrhage should cut the facial fibres before their decussation, and at the same time cut off the descending pyramidal fibres, we would have a paralysis of the face and the limbs on the same side, which would be opposite to the site of the clot. If, however, the facial fibres had already crossed, the decussation of the pyramidal fibres being far below this point, and the clot had destroyed the facial nucleus, the face would be paralyzed on the side of the lesion, while the limbs would be paralyzed on the side opposite to it, i. e., a crossed hemiplegia, or alternate hemiplegia. If the peduncle of the cerebellum is cut we may get a cerebellar type of ataxia. If the motor nucleus, or root-fibres of the 6th, or abducens are destroyed, the movements of the eyes toward the lesion are impossible, but if irritative only, as is generally the fact, they look toward the lesion. The motor and sensory portions of the 5th, are close together, and may be affected together, or separately, and if the motor nucleus is irritated, but not destroyed, trismus may result. The sensory fibres lie above and behind the motor, in what is called the "tegmentum." If the lesion is deep enough, it will affect them, and such losses are frequent, and may predominate. It will be evident then that motion, sensation and cranial-nerve function may be disturbed, irritatively and destructively, and in various combinations, although a motor alternating paralysis is a distinctively pontile paralysis (Starr). The patient has some headache and malaise for a few days, with vertigo and sometimes vomiting. He then suddenly falls into a profound coma, with twitching of the face, or limbs, or both, but rarely with any general convulsions. In other cases consciousness is not disturbed. The face is flushed, there is a fall of temperature, the pulse is irregular, but without much change of rate. Respiration is slowed to from 4-6 per minute, or more often is Cheyne-Stokes. The pupils are pin-point, and do not respond to light; they may be uneven, and the eyeball may be retracted. There may be a strabismus, or a conjugate deviation away from the lesion. There is an evident resemblance to opium poisoning. As the ponto-thalmic fibres may be implicated, there may be forced laughter and crying.

Crus. Hemorrhages in this locality may be downward from the thalamus, or corpus striatum, or may be primary, in which latter case they are small. If diffuse they are quickly fatal, but if circumscribed, and in the ventral portion of the crus, they destroy the motor tracts to the whole of the opposite side of the body. The lower face is also paralyzed, while by destruction of the 3d nerve-nucleus, there is paralysis of all the muscles of the eyeball, except the superior oblique and the external rectus, on the side of the lesion. The 3d nerve issues from the inner side of the crus. Crossed hemiplegia with 3d nerve implication is the "Benedickt Weber" syndrome. Headache is trifling, optic neuritis is unknown, convulsions are rare, and mental changes are slight, and less than in softening. Sensibility is blunted, but hemianesthesia is rare. Increase of sensibility to pain may be present and co-exist with a decrease of ordinary sensibility. There may be spontaneous pains, but less often than in softening. The hemorrhage may go up into the foot of the capsule, and then we shall find hemianesthesia.

Medulla. Hemorrhage into the medulla is extremely rare. It is usually immediately fatal, but if it be small enough to be survived, the symptoms would be those of a bulbar palsy of acute onset, with a paralysis of the limbs and trunk on the opposite side.

The diagnostic mark of all hemorrhages of the crus, pons, and medulla is a crossed paralysis. This means that the paralysis of the body is on the side *opposite* to the lesion, and of the cranial-nerves on the *same* side as the lesion. The level of the lesion will be indicated by the particular nerves implicated. Lesions of the crus *directly* affect the 3d; of the pons the 5th, 6th, 7th, and possibly the 8th, while those of the medulla affect the 9th, 10th, 11th, and 12th.

Differential Diagnosis. Coma is a symptom common to many maladies, and, therefore, in all cases of coma obtain a sample of urine by catheterization, remembering that during the first 12 to 24 hours of an apoplectic attack the urine may, as a direct result, contain albumin, and sugar less often, and to a slight degree. Take the temperature in the rectum, note the respiration, state of the pupils, and of the superficial and deep reflexes. Examine the skull, and obtain all the possible data as to the manner of onset.

Uremic Coma. The condition cannot be certainly diagnosed, while the patient is in coma, by the urine alone, since apoplexy tends to produce an albuminous urine for the first 12 to 24 hours, although there are no casts in the urine of an apoplectic *from the apoplexy;* if present they are from a preexistent nephritis. If there are convulsions present which tend to shift about, they are probably uremic, while if unilateral the cause is probably cerebral apoplexy. The history of previous nephritis would be a guide. Uremic coma comes *after* vomiting, headache and convulsions, while the vomit of apoplexy comes after the coma, and the coma of uremia is not absolute, and the patient is restless, instead of lying like the apoplectic in perfect apathy.

Diabetic Coma. There is a uriniferous (and sweet) odor of the breath found in no other complaint. The coma is not of sudden onset, but is the culmination of a condition of somnolence and headache, anxiety, and sensations of suffocation of some duration. Urinary examination determines the diagnosis more than in uremic coma, since the glycosuria of apoplexy is very slight.

Epileptic Coma. An epileptic attack presents no particular similarity, since there has been a tonic, followed by a series of clonic convulsions, followed by stupor, while the reverse is true in the convulsions of apoplexy; but if this history is unknown, and we find a person in the stuporous stage, which may be unusually prolonged, or replacing the usual attack, we should then examine the tongue for bites, or scars of old bites. Unilateral symptoms are rare, the patient is youthful, arteries are good, there are capillary hemorrhages in the conjunctiva, and on the cheeks. The knee-jerks are abolished in both conditions; exaltation of the deep reflexes may be found for a few moments in both. Apoplexy has been known to occur from the cerebral strain of an epileptic convulsion, and, therefore, is a possible complication.

Focal Epilepsy. This may indicate a cortical hemorrhage, and is common in all diseases of the motor cortex. The attack may resemble an apoplectic attack, and the history alone is a guide, since the paralysis may exactly resemble an apoplexy. In a young person think of multiple sclerosis.

Syncope. This and the two next conditions are barred out if there is a definite one-sided paralysis. In syncope we find a pale face, sighing respiration, consciousness only partially abolished, dilatation of the pupils, preservation of the deep reflexes, and rapid recovery.

Narcotic Poisons. General convulsions never occur. Opium produces a slow respiration, small and thready pulse, cool and blue skin, and a most extreme contraction of the pupils, and the corneal reflex is never lost. Temperature is low, there is no paralysis, and coma is never absolute.

Alcoholic Intoxication. *General* convulsions never occur. There is the odor of alcohol, nature of the vomit is diagnostic, there are motor restlessness, tendency to delirium, absence of a low or unequal temperature. Remember that a man may become intoxicated and rupture a weakened artery, while he is also more liable than a sober man to fracture his skull. In such cases, his being alcoholic does not bar out apoplexy. There is no paralysis, nor are the pupils unequal.

Test for Alcohol. Add a few drops of urine to the following mixture: Solution of Potassium Bichromate 1 part; Sulphuric acid, fortior, 300 parts. The result will be a bright emerald color. (Turner, in Allchin.)

Hysteria. At times it looks very like, but the patient is apt to be young, the face is exempt from paralysis, and betrays the presence of hallucinations. Stigmata of hysteria may be present, and psychic suggestion, or ovarian pressure, may terminate the condition at once. The pulse may be *accelerated*. but is never *retarded;* the pupillary reflex remains, and the deep reflexes also, and there is no Babinski. Temperature is normal, paralysis is flaccid, and there is often anesthesia.

General Paresis. The condition in this disease which simulates apoplexy is a true apoplexy, in the sense that it is a dural hematoma from the rupture of an artery in the meninges. It is much sooner and much more completely recovered from, as the brain is not torn, but only compressed. The knee-jerk is exaggerated, there are facial tremor, speech defect, Argyll-Robertson pupil, and a history of grandiose, or melancholy delusions. There is a history of mental failure.

Fracture of the Skull. In almost all cases there is bleeding from the mouth, nose, or ears. The coma in these cases is due to the rupture of a meningeal vessel. The diagnostic point in hemorrhage from the base of the skull is that the blood does not clot, due to the presence of sodium chloride in the cerebro-spinal fluid with which it is mixed.

Acute Softening from Embolism and Thrombosis. The embolic patient is young, valvular disease is evident, or there is the parturient condition. Coma is shorter or slighter, and the temperature is only slightly disturbed, and if so is elevated and not depressed, and paralysis comes first, and then coma comes on after convulsive movements, which is the reverse of the order in apoplexy. The face is pale, the pulse is small, and the onset is generally gradual, but may be as sudden as apoplexy. There is no unilateral variation of surface temperature.

Thrombosis. The patient is old; or if young has had syphilis; or if an infant has had marasmus, or some similar disease. The onset is often during sleep, and for some time previous there has been some blunting of intellect, confusion of ideas, and of intelligence. Restlessness at night is a very common prodrome. The pulse is irregular, and we find hard radial arteries, and there is a condition of general atheroma. The pupils are even, and react to light; the face is pale. Coma is light, and twitchings show which side of the body is to be affected. Twitchings are common, while convulsions are rare. Coma lasts only for from twelve to twenty-four hours. No *fall* of rectal temperature. Thrombosis usually occurs in sleep, and when blood-pressure is lowered, while apoplexy is the accident following an elevation of blood-pressure. The explanation of the fact that many apoplexies occur in sleep is that the venous return is impeded by position, or by some compression of the veins.

Blood pressure is high in hemorrhage, high in embolism, low in thrombosis. In hemorrhage the retinal vessels are engorged, the disc is cupped, and this is seldom true in thrombosis or embolism.

TREATMENT. The conditions leading to an apoplectic seizure are often discoverable. Increased blood-pressure is most generally the objective symptom, and its cause should be the subject of our inquiry. Arteriosclerosis or arterial degeneration may be obvious, and in all such cases regulation of diet, occupation, and the modification of unfavorable surroundings should be instituted. Occasionally we may be able to reach a patient exhibiting the symptoms of an impending hemorrhage. In such a case Aconite, Belladonna, Veratrum viride, aided by ice-bags to the head, and hot bottles to the feet, and swathing the legs in hot towels may avert the attack, for a time at least. Nitroglycerine in doses of 1/100 gr. may be necessary from the necessity of prompt dilatation of the arterial system. Starr suggests Adrenalin hypodermically, or by rectum. Almost invariably the stage of inception is missed, and we see only the victim of an apoplexy. Can we check the hemorrhage? If it is possible to assure ourselves that it is meningeal, it can be treated surgically. Starr quotes a case where McBurney found the clot, removed it, and the patient recovered. I had a similar case, but although the clot was localized and removed, the result was not so favorable. Attempts have been made to operate on deeper clots, but results are not encouraging. The average case should be left quiet where he falls, with loosened clothing, for some hours, if it is possible. If he is removed, it should be most carefully done, with the head and shoulders slightly raised, avoiding bending the head forward. Apply ice to the head, and heat to the feet, and mustard-leaves perhaps, unless they irritate the patient, and make him indulge in a constant struggle. Then they are worse than nothing. Where the blood-pressure is very high, and the coma very deep, all these measures should be employed. Sinapisms to the nape of the neck stimulate the arteries of the brain to contract, which is the purpose of the ice-bags to the head. Church has suggested the compression of the carotids for forty minutes if the case is seen early. Dawbarn and Thompson have advocated the use of the Spanish windlass on the limbs, close to the body for one hour, just tight enough to obstruct the venous return. If the blood pressure is high, and the coma profound, bleeding to the amount of ten or twelve ounces is advisable; the same result may be produced to a less extent by withdrawing a moderate amount of the cerebro-spinal fluid by lumbar puncture.

Remedies here are Belladonna, in sthenic cases, but Aconite if there is more excitement, and less coma. If the coma is deep Opium is preferable, and if the hardness of the pulse is conspicuous Veratrum viride may be given in potency, or in drop doses of the tincture every hour until the pulse is softer. With an irregular and feeble heart action, use Glonoin in the first dilution. When the condition is one of apathy, and the hemorrhage seems to be a continuing one, as is often the case, Hamamelis in tincture has seemed valuable. In all cases empty a full bladder, and clear out the bowels. On account of the difficulty of swallowing, medicine should be given drop by drop on the tongue, and not by the teaspoonful. A good purgative at this stage is Croton Oil, 2 drops on butter, and placed on the tongue. Later the bowels should be kept clear by enemas. In finally disposing the patient after the initial period, no shock or jarring should be allowed. Clothing should be ripped along the seams, and not pulled off, unless it can be removed with very slight movement of the patient.

Whether, or not, position reduces blood-pressure, it seems wise to place the person on the paralyzed side, since the saliva thus runs more easily out of the mouth. Keep the mouth closed by favorable position as far as possible, since the drying of the mouth is thus mitigated. Do not feed, since the patient can neither swallow, nor digest, and administer water by wetting the mouth and the lips. Keep the room cool, dark, and quiet, and do not allow the solicitude of the friends to annoy the patient.

When consciousness returns, administer plain and unstimulating food in small quantities, semi-fluid is the best form, but never *raw* milk. There should be no stimulants, but fluids of a refreshing character in moderate amounts. Easily digestible solid food may be given at the end of a week. The patient should have his position changed frequently, and he should be sponged with alcohol and water, but in all these measures he should be warned against effort on his part. These changes and cleanliness, and keeping the skin dry, is the best method of combating bedsores. The bladder should be emptied by a catheter, and the bowels should be kept open. This is the period of irritation, and Bryonia in the lower potencies is of value, but Belladonna, Apis, Hellebore, or Cicuta may be more specifically indicated. If the only symptom of this is headache, indicating implication of the meninges, we should then consider Bryonia still the better remedy if there is great tenderness of the scalp, but if this is not the fact Melilotus is preferable. About the eighth day the movements of the limbs indicate that compression is reduced, and Ignatia, or Nux vomica 3x are of service.

Drugs have a limited sphere in the treatment of this accident at any stage, and in the later ones massage and faradic electricity should be faithfully used to develope to the full the wasted muscles. Contractures are possibly benefitted by them, and the patient should be encouraged to attempt movements which are imperfectly accomplished. Contractures of the hands should be antagonized by gripping motions, and the weak ankles should be moved to and fro against resistance. Muscle-beaters can be used with advantage on the contracted muscles, and warm baths followed by oil-rubs will be found helpful.

Electrical Treatment. In predicting the value of electricity in a given case of apoplexy, it must be remembered that this agent will not restore nerve-cells which have been destroyed, nor will it replace nerve-fibres whose nutritional source was destroyed when the cell died. It will stimulate cells which were injured but are still capable of function, it will preserve the volume and integrity of muscles which are only awaiting the recovery of the part of the cortex from which they are innervated. Its value in accelerating the absorption of a clot is rather doubtful. If it can affect the clot favorably, it will do this under the influence of a galvanic current passed through the skull so that the clot lies in the field of force. If it does produce any effect, it will be manifest within three weeks, and it should not be employed until three or four weeks have passed since the attack. The anode should be placed over the lesion, and the cathode at various points on the opposite side of the head. A more powerful method is to place both electrodes upon the same side, but the anode should be kept over the lesion. General galvanization to the spine will increase the nutrition of the spine, and secondarily of the whole body. At another sitting, so as to avoid fatiguing the patient, the muscles should be exercised by placing the anode upon the back, and the cathode over the motor points of each affected muscle, with an interrupted current. This fatigue is shown by a decreased motor response to the current. The proper treatment is outlined in the article upon Spastic Paraplegia.

Aphasia is generally due to the destruction of the cells in some one of the speech centers, and is not amenable to treatment. If the cause is an ischemia, or the temporary shock to the cerebral circulation, it will be benefitted by placing the anode over the posterior part of the third frontal convolution, and the other on the right side of the larynx, passing a galvanic current for two or three minutes. Then place the anode on the back of the neck, and the cathode, using a small Erb's electrode, on the motor-points of the external and inferior laryngeal nerves, pressing the electrode firmly into the tissues. The static breeze is valuable as a general nervous stimulant, and strong sparks over the dorsal spine seem to improve locomotion, and over the cervical plexus are beneficial to movements of the arms. Anesthesias are also relieved by the static breeze and the faradic brush. The galvanic anode relieves pain in the muscles at times.

In the later stages, after the patient's condition has become a chronic one, Nux vomica in the lower dilutions seems to accelerate recovery more than any other drug, but if the case comes to a halt under its use, Baryta carb., Causticum, or Hepar may be found of use. Strychnia phos. 3x, or in material doses, or Zinc phos. 2x, will accelerate improvement.

SOFTENING OF THE BRAIN.

THROMBOSIS AND EMBOLISM. ENCEPHALOMALACIA.

DEFINITION. It is a condition of necrosis of the brain substance, which arises from the occlusion of the blood-vessels. In the acute form it is caused by thrombosis or embolism of the arteries, and the initial symptoms closely resemble those of cerebral hemorrhage. There is a chronic form which depends more directly upon structural changes in the blood-vessels, usually of the smaller arteries, and capillaries, which by slight attacks, or a continuing process produces motor, sensory, or psychic losses, to a variable extent.

SYMPTOMS OF A TYPICAL CASE. If the cause is the occlusion of one of the arteries by a thrombus, the patient will rulably suffer for some days before the attack with insomnia, restlessness, headache, and confusion of mind. Some grade of paralysis will then come on, probably succeeded by some minor grade of unconsciousness, which paralysis initially is widespread, but after a week or two is reduced to very narrow limits. Aphasia is a very common phenomenon. An embolus produces as sudden a stroke as a hemorrhage, and premonitory symptoms are lacking, but the later history is like that of thrombosis. In the case of both thrombosis and embolism the pulse will be small and rapid, the temperature will be normal, or elevated, the face will be pale, and not red. In the chronic type, depending largely upon arterial and capillary degenerative changes, unconsciousness will be rare, but some disturbance of consciousness will be the rule; there will be bodily restlessness, single muscles and members will be paralyzed, or the permanent losses will be weakness and clumsiness of movement only, and almost without exception there will be some grade of mental impairment.

DIFFERENTIAL DIAGNOSIS. It is to be diagnosed from Cerebral Hemorrhage, Hematoma of the Dura, Tumor and Abscess of the Brain, and Encephalitis.

AGE. It is a possibility at any stage of life. The infant, after exhausting diarrheas, is apt to develope a thrombus in the vessels of the brain, and this may also be a result of acute infections. This possibility is conspicuous throughout childhood and adolescence, the period when persons are most vulnerable to the acute infections. Old age is peculiarly the arena for thrombosis, because the blood has a higher viscosity, the arterial intima is liable to have become roughened, and fatigue is frequently of such a grade as to slow the blood-stream to a dangerous degree. Syphilis may cause a thrombosis at any age of life.

During the prime of life embolism is more common than thrombosis, as the male is then exposed to muscular strains, any one of which may tear off a vegetation which will become an embolus, while the female is at the same time encountering the dangers of the puerperium.

SEX. Males are rather more liable to thrombosis, while females suffer more often from embolism.

ETIOLOGY. A thrombus is a plug occluding an artery, or a sinus, which has been developed from the fibrous elements of the blood at the point where the occlusion takes place. An embolus is a plug in an artery composed of tissue of various sorts, and is brought from a distance to the point where it is occluding the artery. A thrombus may arise from any condition which degenerates the coats of an artery. Syphilis has this effect pre-eminently, and in cases occurring under the fortieth year it may almost certainly be predicted as the cause. The later stages of nephritis, diabetes, alcoholism, intoxications by the poisonous metals and illuminating gas are competent for its production. Poisoning by illuminating gas weakens the muscle of the heart, and produces thrombosis, especially in the artery leading to the lenticular nuclei, since that artery makes a right-angled bend. It is, therefore, a characteristic of gas poisoning that it produces softening of these two nuclei. This is the cause of death in the fatal cases, and of motor palsy in those who survive. The pathological processes giving rise to general paresis, progressive bulbar palsy, and multiple sclerosis, entail this probability, and it frequently occurs from the atheromatous and arteriosclerotic changes incident to old age. In the earlier stages of these processes the intima is in an abnormal condition of roughness, and the lumen is narrowed, and only the added condition of an increase in the coagulability of the blood, or a slowing of the circulation, or both at the same time, are necessary to ensure the formation of a thrombus; it is therefore apt to occur during the sleep of an aged person. Infancy is prone to diseases of exhaustion, which may also diminish the fluidity of the blood, or to acute infections which affect the arterial walls, as well as the composition of the blood. Phthisis or the puerperium both tend to produce some grade of endarteritis, and the coagulability of the blood is at the same time increased, hence the accident is not uncommon under those conditions. Diseases affecting the myocardium may contribute to the formation of thrombi by weakening the force of the blood-stream, while at the same time the endothelium of the arteries is in an irritable state, and the condition of the blood is favorable to coagulation (as in gas poisoning). It has been said that agglutination precedes coagulation, and not the contrary as was supposed formerly.

A new view of the etiology of thrombosis has been advanced by Howell. Coagulation requires the presence of thrombin and fibrogen together. There is an inhibiting substance in the blood termed antithrombin, but if this becomes neutralized coagulation will occur. There is normally in the blood-plasma, and also in the so-called peptone plasma, a substance which has the power of inhibiting the action of thrombin on fibrinogen. This substance so closely resembles antithrombin in its general properties that it is fair to call it by this name. Commercially, there is such a substance known as hirudin, obtained from the heads of leeches. There is an element in the blood which exists in almost every tissue of the body, which neutralizes the antithrombin, since clot formation is usually a safety measure. For instance, if fibrinogen, thrombin, and peptone plasma be mixed together, there will be no clotting, but if an extract of thymus and spleen be added, coagulation will occur, showing that in these tissues is a substance capable of acting as an antibody to antithrombin (Tilny). This new theory points out the probable mechanism, and suggests a possible method of prevention when the technical difficulties are surmounted.

As a practical clinical fact, embolism usually occurs in two classes of persons, in the prime of life, from two definite conditions. A man who has previously suffered from rheumatic endocarditis is exposed to a severe acute shock, physical or mental, or lifts a great weight. He tears a fragment from the vegetations encrusting his mitral valve. It flows in the blood-stream until it reaches an artery of the brain which is too small to allow its further passage. A woman is confined, and during the early days of uterine involution a clot from one of the uterine vessels breaks loose, is carried up to the heart, and thence to the brain, where it occludes one of the vessels, or she has a phlebitis, and a clot is carried from the saphenous vein to the brain in the same manner. Emboli may result from any disease in which the endocardium has been the site of inflammatory action, and, therefore, it may occur during or after typhoid, pneumonia, diphtheria, or the puerperal state. Tuberculous disease of the lung may cause a local thrombosis; pieces of tissue from the affected area may be washed away into the circulation, and finally lodge in the brain. Calcareous particles from an atheromatous artery, filiaria, or hydatids may furnish the material for an embolus. It has resulted from lacerated wounds, has come from the degenerating areas in carcinoma, and after extensive burns fat emboli are likely to occur. Dr. Quervain reports fat embolism following a bloodless reduction of an anchylosed joint, and Crusen another from abdominal section.

PATHOLOGY. If a plug forms in a small terminal artery of the cortex, we shall find a small bloodless area, surrounded by a larger one which is congested. There is an exudation of serum, and an invasion of leucocytes by the third day. The prevailing color is at first white, but this soon becomes red, on account of the reduction of the serum, and the presence of the extravasated blood corpuscles. The leucocytes are now filling themselves with the disintegrated blood-cells, and the debris of the tissues, and the parts become yellow, which condition is reached in about six weeks. In about sixty days, the leucocytes will have cleared the area of all dead tissue. When, therefore, we speak of white, red, or yellow softening, we are speaking of different stages of a single process. If the spot was a very small one the process will terminate in the formation of a minute mass of neuroglia tissue (a scar), but if it is larger there will be a cavity, or a cyst, filled with clear fluid. The part is dead, functionally speaking, since nerve-cells die in about forty-eight hours, if deprived of their blood-supply. This is the condition which obtains in the small occlusions which are so common in alcoholism, syphilis, general paresis, and the changes of senility.

In the case of a thrombosis, or embolus, we find, instead of a more or less circular spot, an infarct, in which the subsequent process is the same as has been just described. Thrombi tend to form in the large central arteries, while emboli affect those of smaller calibre. They also are generally situated at the bifurcation of arteries, and may grow backward by accretion from the blood-stream, or may subsequently become perforated, so that the area again is nourished to some proportion of its former supply. An embolism not infrequently follows the disintegration of a thrombus, and thrombosis may succeed embolism. If the embolism occurs from a fragment from the site of an infection, as ulcerative endocarditis for instance, we shall find that the infarct is infected, and an encephalitis will be the result. The clinical results differ according to the size of the occluded arteries. If a large artery is closed, a large area of the brain is affected, and the initial shock is great, but from anastomosis a new avenue of supply is soon developed, and the paralyses will be transient. The small arteries are terminal, and there is no anastomosis; therefore, while the initial shock is quite slight, the resulting paralysis, such as it is, will be permanent.

Therefore the degree of paralysis resulting from embolism is more likely to be permanent than that from thrombosis, and while anastomosis is good at the cortex, it is absent in the central ganglia.

LOCALIZATION. The most frequent site for softening is in the basal ganglia, and the neighboring internal capsule. The middle cerebral arteries and their branches are the most frequent site for embolism, and the arteries of the base for thrombosis. After the lenticular nucleus, with the internal capsule, next in frequency come the caudate nucleus, optic thalamus, and the adjacent parts of the centrum ovale, and corona radiata. It attacks the anterior part of the centrum ovale more often than the posterior, or occipital. The tempero-sphenoidal lobe is often softened, but is rarely the site of hemorrhage. Lastly we find the fewest cases affecting the pons, cerebellum, midbrain and corpus callosum. Basal cases are almost always syphilitic, and show a successive implication of the smaller arteries leading to mental deterioration, dysarthria, headaches, vertigo, and changes in sensation.

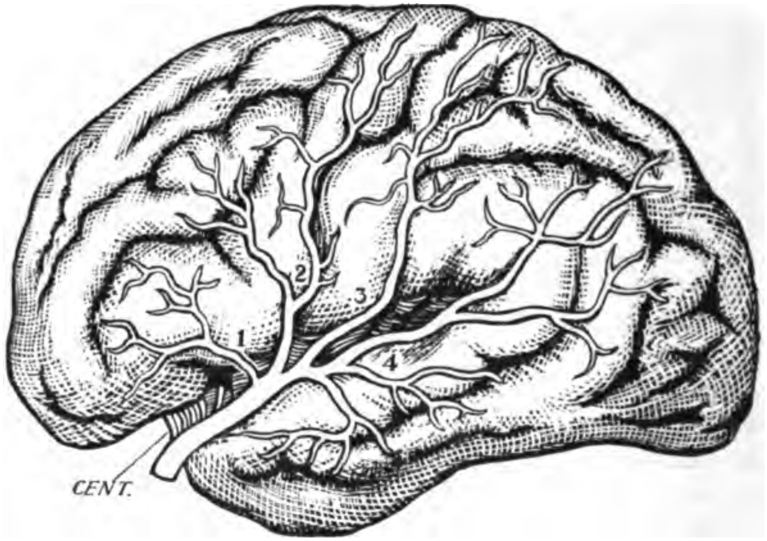


FIGURE 82. THE DISTRIBUTION OF THE MIDDLE CEREBRAL ARTERY.
1. Subfrontal branch. 2. Precentral. 3. Postcentral. 4. Parieto-temporal, and Temporal. *Cent.* Central branches. (After Duret.)

SYMPTOMATOLOGY. When the process is chronic, depending upon the gradual occlusion of the arteries of the circle of Willis, then, and then only, we may have a bilateral paralysis. In senility the atheromatous process may finally lead to occlusion, and the same result is seen in senile dementia, senile epilepsy (Spratling), and alcoholism. In these latter it is apt to affect the gray matter at the base of the frontal and temporal lobes (Dana). In all these cases the patient will show a variable amount of shock proportionate to the suddenness of the occlusion, and the size of the vessel. Unconsciousness is not the rule; there is rather a clouding of the faculties, headaches and vertigo, and a paresis of single parts, or even of single muscles. Spasms, twitchings and rigidities are features when the cortical areas are affected; emotionalism, dysarthria, and changes of sensation when the central ganglia are attacked. When the larger arteries are affected we may get a hemiplegia in stages, and it may simulate a brain tumor in some cases from the fact that there may be optic neuritis and atrophy. After a certain amount of progress there may be no further changes.

Thrombosis differs from both multiple hemorrhage and embolism in having a premonitory stage which may be days, or even weeks, in duration. The patient is subject to headache, and reduced mental ability, and, at times, irritability. The patient is apt to be petulant, and is constantly seeking some cause of his unrest in his environment. A patient of my own, under the influence of a forming thrombus, changed his room in a hotel four times on one night, and had his bed made and re-made a half-dozen times. There is apt to be pain in the parts about to be paralyzed. When occlusion is completed, we shall find one of two classes of symptoms. If the occluded artery is large the patient will be found, generally after sleep, to be clouded in mentality but while difficult to arouse, is not unconscious; at times consciousness will be lost, but rulably it is neither as deep, nor as prolonged as in cerebral hemorrhage. He will more often be paretic, that is weakened muscularly, and incoördinate, but not acually paralyzed, and he is often aphasic. This condition will endure for a few days, when it will improve, and the aphasia generally shows improvement, and in a week or two disappears, except that some degree of hesitation in speech remains. When the patient is tired, or emotionally overtaxed, he is liable to a temporary renewal of the aphasia. Initially there is a rise of temperature of one or two degrees, and the pulse is rapid and small. If the thrombi are multiple, and small and widespread, a common form in the aged, there will be no initial shock, but the result will be an increasing feebleness, accented on the days when a new vessel is occluded. The patient is progressively more and more feeble in mind and body, the muscles lose their tone and there is a marked inco-ordination of movement, so that the patient's movements are clumsy and weak, resulting in the "short-step" progression of the aged.

Embolism differs from the two preceding causes of softening in the fact that it is as abrupt as massive cerebral hemorrhage, and has none of the premonitory symptoms so usual in thrombosis. There may be all stages of mental confusion, or coma, and complete unconsciousness, but Hippel reports two cases, confirmed by autopsy, where there was no coma, nor unconsciousness. Spasms or epileptiform convulsions are a pronounced characteristic of most cases, and the mental confusion may alternate with delirium. The temperature is elevated, as in thrombosis, and the pulse, here also, is weak and rapid.

In both thrombosis and embolism the patient is apt to suffer extremely from muscular cramps, and all sorts of paresthesias. There will be stinging pains in muscles and skin, and burning and other unpleasant sensations, which may be so severe as to prevent sleep. While the paralysis in both conditions is initially widespread, it will improve far more than we should expect, and much more rapidly, yet it must be remembered that it is not a negligable condition, and that which persists more than three weeks is liable to represent a permanent loss, since the collateral circulation, upon which cure depends, is completely developed within a few days. The muscles lose their tone from atrophic conditions, and this is the condition in all sorts of tissues, and may be severe enough in grade to produce bedsores. The muscles of respiration and of the heart may be affected, and their implication indicates extreme danger to life.

SPECIAL FORMS OF SOFTENING.

Pons. Softening in the pons is most liable to be in the ventral part. near the raphe, and next to this, in the dorsal part. The basilar artery or its transverse branches are involved. Lesions of the pons alone are rarer than those involving also the medulla, especially of its upper part, because the basilar is not as often plugged as are the vertebrals and their branches. The prodromal symptoms are not as prolonged. In a typical attack due to plugging of the basilar we get softening of the deeper ventral portion of the pons. The patient suffers from a hemiplegia with some paresis on the side opposite the hemiplegia. There is great exaggeration of the reflexes, and twitchings, spasms, and coarse, irregular, jerky movements of the limbs, but no convulsions. There is usually anesthesia and ataxia on the side of the lesion, and we get a dissociated anesthesia. There is often forced laughter and weeping, dysarthria may occur; forced movements are not common. When the lesion is high up, and involves some of the transverse branches of the basilar, we get oculomotor palsies, and spasmodic disorders on the side of the lesion, and paralyses and anesthesia on the opposite side. When lower down we get 5th, and 7th nerve palsies on one side, and hemiplegia on the other. If in the basilar lesion the injury is large and severe, the patient developes disorders of deglutition and speech from extension of the process, and from paralysis of the bulbar centers. No great disturbance of temperature occurs, nor are there marked pupillary symptoms. In the smaller lesions, due to involvement of the transverse branches, recovery may occur, leaving the patient with a crossed paralysis.

The medulla is rarely the site for softening, but more often than for hemorrhage. It is apt to occur between the ages of 30 and 50, and more often in males. It is due to syphilis, nephritis, and the arterial changes due to it and to alcoholism. Gordonier reported three cases, and states that the artery involved is the posterior inferior cerebellar branch of the vertebral. It may come on slowly with general arteriosclerotic symptoms, superimposed upon which are symptoms due to the irritation or destruction of the cranial nerve-nuclei and their root-fibres, the tract of Gowers, direct cerebellar tract, the descending vestibular tract, and the cochlear nerve-roots, the dorsal and ventral auditory nuclei, the cerebello-olivary tract, the nucleus ambiguus, or combined motor nucleus of the glosso-pharyngeal and pneumogastric nerves, the vasomotor and sympathetic fibres. Often its onset is sudden, and the symptom-complex is characteristic. Consciousness is neither lost nor disturbed, vertigo is marked and persistent, with nausea and vomiting; deafness on the side of the lesion, absolute loss of the power of swallowing, owing to loss of the power of the muscles of deglutition on the side of the lesion. The voice is low and hoarse. There is staggering toward the same side as the lesion, often striking objects on that side, and the gait closely resembles that of cerebellar disease, i. e., drunken gait. There is complete analgesia and thermesthesia on the arm, trunk, and leg of the side opposite the lesion, and in one case of the face of this side also. There is absolute preservation of the tactile sense on the whole of the side of the lesion. In one case there was a diminution, or loss of the pain and temperature sense in the distribution of the right trigeminal nerve. There was impairment of the sense of pain and temperature in the larnyx, while the sense of touch was retained. The pupil was contracted, there was ptosis, narrowing of the palpebral fissure, retraction of the eyeball upon the side of the lesion, and also a lack of sweating on that side of the face and neck. The motor power was normal, and the reflexes showed no characteristic change. In one case out of three observed there was a temporary facial paralysis on the side of the lesion. There will be no permanent paralysis in the domain of the facial and the hypoglossal, unless the thrombus extends into the vertebral. Pulse will be slow if the vagus is irritated, but rapid if it is paralyzed.

Thrombi and emboli are rare in the cerebellum, and in view of the considerable anastomosis they will be small in area. The only difference between softening and hemorrhage is that the symptoms will be more gradual in the developement of softening. The prodromes are like hemorrhage in the cerebrum, pain in the occipital region, persistent vomiting. If the patient does not die immediately after loss of consciousness, he remains comatose. When consciousness has been regained, the striking feature will be the absence of hemiplegia, which is so constant a symptom in cerebral strokes. A hemiplegia has been seen on the side of the lesion, and affects all the muscles of both arm and leg, while in cerebral cases only certain groups are affected. Moreover, there are no contractures, reflexes are exaggerated, no rigidity, no ankle clonus, no toe phenomena; after recovering there is no paralysis; but a weakness, so that the patient cannot even sit in a chair.

Prognosis. Thrombi from acute infections in infancy and youth, or from the exhausting diseases, carry no tendency to recurrence. When thrombosis occurs in middle life, it is in all probability syphilitic, and may respond to treatment. The thrombosis of the aged, or the nephritic, diabetic, alcoholic, epileptic, or paretic individual tends to recurrence, since it is incidental to a progressive deterioration of the vascular system. Thrombosis does not hold out a good prospect for recovery when due to these conditions, because the vessels are also diseased which should furnish the collateral circulation. Embolism is not likely to recur, and the age of the patients gives greater promise of recuperation. In the prognosis of a specific attack, the persistence of coma for several days to even a week need not be a cause for discouragement, but if it is very deep it is of bad omen. Many cases of embolism die in the attack, as do some cases of thrombosis, but the small multiple occlusions mark stages in gradual deterioration which may go on for years. If a case of embolism revives after a day or two, the prognosis for life is good, but in thrombosis there is a second period of great danger at about the tenth day from the secondary softening. Bulbar symptoms in either thrombosis or embolism indicate a probably fatal issue. Some cases of embolism recover, resulting from establishment of collateral circulation, but an aneurysm may develope on the cardiac side of the occlusion, and sudden death occur from apoplexy at a later period. If the paralysis does not recede in two or three weeks, we cannot predict recovery, since the re-establishment of collateral circulation is accomplished in a few days.

Diagnosis. The diagnosis between cerebral hemorrhage, thrombosis and embolism is very often impossible. G. Burke and J. Nuzum report a case of a woman sixty-six years, with coma, the presence of high blood pressure with high tension pulse, conjugate deviation of the eyes and head, elevated temperature, albumin in the urine; all these findings seemed to make the diagnosis of cerebral hemorrhage easy. However, post-mortem examination showed brain softening due to thrombosis of the right middle cerebral artery, thus showing that the differential diagnosis is in many cases impossible. The optic discs are congested in hemorrhage, and are normal in thrombosis and embolism.

The general distinction between hemorrhage and thrombosis is that the latter condition has had the prodromal stage of several hours or days; that the patient is conscious, or not in absolute coma; that he has a pale face instead of a congested one; that his pulse is small and feeble, and rapid, instead of slow and full; that his temperature shows very little change from the normal, or is elevated instead of depressed. Later: that he soon recovers consciousness, if it has been lost; that a wide-spread paralysis shows a very rapid diminution in area. While the stroke of embolism is sudden, even more certainly so than in hemorrhage, initial convulsions are more common, and the general symptoms are like those of thrombosis. There are special distinctions also in the difference of causative conditions, as has just been detailed. Brain symptoms, accompanied by a bloody urine and pain in the lumbar region, and an enlargement of the spleen, denote infarcts in those localities, and indicate that the brain condition rests also upon cerebral embolism. In hemorrhage the age is over forty, there is a fall of temperature, and a rapid and progressive improvement is seen soon after the onset. There is blood in the cerebro-spinal fluid, there is congestion of the retinal arteries, and the disc is cupped and hazy. In favor of softening there is a rise of temperature, incomplete hemiplegia, and the presence of cardiac lesions. Convulsions are more frequent in softening than in hemorrhage, especially if they are unilateral, and when the cortex is affected. Aphasia with a mild grade of hemiplegia is almost always due to softening; if it is persistent it is certainly from softening. Hemianopsia suggests softening, as does a repetition of apoplectic attacks. Embolism is more apt to occur on the left side than on the right; the middle cerebral arteries and their ganglia are the usual site for embolism, and the arteries at the base for thrombosis.

DIFFERENTIAL DIAGNOSIS. Hematoma of the Dura. (*Hemorrhagic Internal Pachymeningitis.*) This is generally bilateral, but not equal, on both sides of the body. Marked by headache, vertigo, a tendency to somnolence, and some grade of paresis. More often affecting the limbs than the face. The pupil on one side is apt to be contracted. Spastic contraction of the limbs, and conjugate deviation of the eyes, sometimes. Occasional convulsions, which may be unilateral, or general. Intervals of health, somewhat disturbed by brain-symptoms. Very chronic. Generally preceded by an accident, or precursory pressure-symptoms.

Tumor. There has been a history of a peculiarly excruciating headache, with nausea, and probably vomiting of a projectile type. There is typically a double choked disc, or the succeeding optic atrophy. The motor changes are paresis, or paralysis of a limb, or part of a limb, much more often simple localized spasticity, with jerking and twitching; never a hemiplegia. There is usually no mental change, but at times we find some loss of acuteness, or a sleepiness. Above all, there is a history of slow evolution.

Encephalitis may arise from an infected thrombus, but if primary there is the history or presence of a focus of infection. Somnolence is marked, there are rapid rise of temperature and convulsions, ending in death.

Abscess. There is the history of an infective injury, or purulent disease of the ear, or nose, or the antecedent presence of an embolus from some infected area, especially in the lungs. There are general cerebral symptoms, with a subnormal temperature, or fever and chills, the pulse is slow and irregular, and there is leucocytosis, especially polymorphonuclears.

Treatment. This is in general like that of hemorrhage, except that the patient should be placed horizontally, there should be no letting of blood, nor immediate catharsis. The bladder should be emptied at once, and carefully watched afterward, but the bowels should be left alone until they can be relieved by an ordinary enema. Most authorities advise the prompt use of cardiac stimulation, in view of the weakness of the circulation, but I think that it is obvious that this is theoretically wrong, and I am convinced from experience that it is clinically detrimental.

LATE TRAUMATIC APOPLEXY. SPÄT APOPLEXIE OF BOLLINGER.

Definition. This is a type of apoplexy, always traumatic, where after the immediate shock of the injury has passed away, the patient has a period of functional competency. This period may be entirely without symptoms, or may be marked by evidence of a disturbance of the brain, of a more or less severe type. The duration of this period of delay may be from two or three days to six weeks, after which there is a cerebral hemorrhage, with unconsciousness, and followed by the amount of paralysis determined by the locality of the hemorrhage.

Pathology. This accident does not occur to a person with normal arteries, but the diseased condition may not have been evident. The immediate effect of the accident, according to Bollinger, is to cause an unequal distribution of the cerebro-spinal fluid, which induces local softening. In his cases, which were fatal, the hemorrhage took place in the neighborhood of the fourth ventricle, and the fissure of Sylvius (Bailey). I have had an intimate knowledge of three cases, but without autopsy, and none of them appeared to be ventricular, but in all probability were capsular. The pathology of all these cases appeared to be an aneurismal dilatation of an atheromatous artery by the sudden rise of blood-pressure from the fright and shock of the accident, and the apoplexy eventuated on the rupture of this aneurism a few days later.

Symptoms of Typical Cases. A man of forty-six, of good habits, aside from excessive smoking, was thrown violently to the floor of a railway carriage, by the shock of a collision, although holding on by two straps at the time. Aside from bruises and shock, he was apparently uninjured, was able to walk a mile, eat dinner, and be about the streets until bedtime. He took a railway journey that night without accident. The next day he attended to business, but with an increasing pain in the head. The next day he was able to work less, as the headache became intense. He took another night-trip, when his head ached so badly that he struck himself violently on the nose three or four times, in an attempt to make it bleed, and thus relieve his head, but no bleeding resulted. On his arrival at his destination, he was able to walk, but had an attack of vertigo while waiting to register at his hotel. Recovering in a few moments, he boarded a car, but soon slid from the seat in unconsciousness, and has since suffered from a complete left hemiplegia. Another gentleman, of about the same age and habits, tripped on attempting to enter his motor, and fell heavily on the pavement, but did *not* strike his head. He was able to continue his activities for about a week, when he suddenly became hemiplegic. Another gentleman of athletic tastes, and about the same age, was thrown heavily, while attempting to mount a restive horse. He was very much shocked, but not definitely injured, so far as could be seen. After a short delay, he mounted and rode the horse, as he had intended. After about a week of reasonable activities, he let a paper which he was reading slip out of his hand, became unconscious, and is now hemiplegic. These two latter cases both consulted a very competent physician very soon after the accidents, and he could find no reason to consider them subjects for medical treatment. In view of these facts, it should be the rule to consider the slightest disturbance of function traceable to the brain, as an evidence of organic injury, if an accident has shortly preceded its appearance.

SINUS THROMBOSIS.

Definition. It is an occlusion of the sinuses of the cranium by a coagulum, and may be primary or secondary.

Anatomical Consideration. The transverse sinuses, and the inferior petrosal, empty into the internal jugular vein, as well as all those centering at the Torcular Herophili. The external cranial veins, the auricular and cervical also, form the external jugular.

The internal nasal veins communicate with the superior longitudinal sinus. The external cranial veins pass into the transverse sinus through the parietal foramen. The ophthalmic veins run into the anterior portion of the cavernous sinus. The veins of the middle ear empty into the lateral sinus.

Etiology. The great proportion of thrombosis of the sinuses is secondary.

Secondary Thrombosis. Infections in the vicinity of a sinus may induce a phlebitis of the sinus, or it may be an extension from a similar process in a tributary vein. The most common source is a suppurative process in the middle-ear, or in the bony cells communicating with it. The lateral sinus is the one affected by such a process. Infected wounds, or abscesses of the scalp, may infect any of the sinuses lying near the margin of the skull, the superior longitudinal being most often the one implicated. From similar processes in the orbit, nose, and face, the inferior longitudinal, or inferior petrosal, or cavernous become the site of thrombosis.

Primary thrombosis is due to a combination of the reduction of the rate of the blood current, and an increase in the coagulability of the blood. It arises also from a decrease in the quantity of the blood mass, but entails one other condition, since it is not probable that a decrease in an actual quantity would be unaccompanied by some change in its consistency. It is an incident of infancy when exhausting disease is present, and is then called marantic. Owing to the stage of life the heart does not retain its power, and the circulation is slower than normal, and an exhausting diarrhea has at the same time increased the coagulability of the blood. Old age presents the same favoring conditions, and, in addition, there is generally some degree of atheroma of all the blood-vessels. Exhausting diseases, such as the infectious fevers, and the cachexiæ, as an unusual result, from a weakened heart, coagulable blood, and a degenerated endothelium may produce this condition in persons of any age. Chlorosis has caused it. The usual site is the superior longitudinal while it is rare in the lateral, and very uncommon in the cavernous sinus. There may be a primary infection of a sinus, or thrombosis may arise from compression.

Pathology. The vulnerability of the sinuses rests upon the fact that their lumen is crossed by numerous trabeculæ. Pacchionian bodies project into them as the years increase, veins empty into them against the direction of the blood-current, and the direction of flow is often against gravity. In primary cases the coagulum is at first red and soft, while in later stages it is whiter and harder. In infections and secondary cases it is varied in color, with collections of pus, and abscesses may form in the wall in the vicinity. Thrombosis of one sinus may extend to the analogous one on the other side of the cranium, or for some distance into the contributing or emissary veins. In any case the contributory veins of the cranium and brain are congested and tortuous, and that part of the brain and meninges is in a state of congestion. This may give rise to capillary hemorrhages, infarcts, areas of softening, and abscesses. The blocking of venous drainage may induce hydrocephalus.

Symptomatology. The causative conditions generally furnish most of the symptoms, and may have been previously so severe that the new condition adds few new elements to the symptom-complex. The general symptoms, when noticeable, are these: in primary cases the following symptoms may be present: there are prostration, severe headache, vomiting and stupor; then delirium with unilateral, or generalized convulsions, and paralysis, and more rarely, incoördination, contracture, tremor, etc. There may be rigidity, nuchal and general; hemiplegia, paralyses of the cranial nerves, especially ocular. The temperature is normal or elevated to 108°, going up by bounds; pulse normal or retarded, rising later; respiration similar. These symptoms are like brain abscess or meningitis with softening.

Secondary: A thrombosis of the lateral sinus, which is not septic, and is discrete may give rise to no symptoms beyond headache and fever. Generally the symptoms are characteristic, because an abscess outside of the sinus and the dura is generally the transmitting medium. The symptoms are inflammation of the bones, sub-periosteal abscess, phlegmon behind the mastoid process, especially around the mastoid foramen, and the neighboring part of the occiput, also on the mastoid process itself. We find pain on pressure and percussion of this region, limitation of movements of the head, then the general symptoms mentioned in the primary cases, plus optic neuritis, and choked disc. If emboli break off from the infected thrombus, we get chills, fall of temperature, followed by a high temperature, and all the symptoms of sepsis.

Localizing Diagnosis. Sinuses in the anterior fossa of the skull, if occluded, produce dilated frontal veins, cyanosis of frontal and orbital region, swelling of eyelids, protrusion of the eyeball, maybe blindness.

Transverse Sinus; swelling behind the ear, and of the veins of the skin, but the jugular is also involved in most cases.

Cavernous Thrombosis may be suspected, if, after an attack which gives ophthalmic vein symptoms, we get an extension to the other side.

Occulsion of the Lateral Sinus involves the jugular vein most often. Systemic symptoms, chills, fever even to 108° with stiff neck, vertigo, vomiting, retarded pulse, irritability, sensitiveness of spinous processes, all meningeal symptoms. Symptoms in the lower extremities have also been observed, Westphal's sign, contractures, paraparesis, etc.

The symptom of Thrombosis in children, to diagnose between it and the Hydrocephaloid of Marshall Hall, is that the fontanelle is elevated, while in hydrocephaloid it is depressed.

Marantic almost always in the posterior part of the longitudinal sinuses, and may also be found in the transverse sinuses.

Otitic. Mostly in the transverse at the sigmoid fossa; at times in the cavernous, and the inferior and superior petrosal.

Traumatic in the superficial sinuses. Injuries to eyeball, face, and nose in the cavernous.

DIAGNOSIS. While the primary thrombi may result in areas of softening, and the patient may survive, and even attain a fair degree of health, the septic thrombi are fatal within ten days unless they are successfully operated, which is possible only when the thrombus is in the lateral sinuses.

TREATMENT. In the case where it affects the lateral sinuses, surgery is the only treatment, and is often curative. It is rarely of any avail in the longitudinal sinuses, and the internal are beyond our reach. In primary cases the patient should be kept in a recumbent position, and if the thrombus is in the longitudinal sinus, the neck should not be flexed. In the marantic cases the causative condition engages all our attention, but in any case ice-bags on the head will be needed. Attention should be given to the eliminating functions of the bowels, kidneys, and skin, and a supporting diet with stimulants should be administered.

THERAPEUTICS. There are slight grounds for much hope from the action of remedies, but Lachesis, Naja, Crotalus and Arsenicum have been recommended. The basis for their use rests upon the clinical fact that recoveries have taken place in the experience of the author which seemed to have some probable relation to the action of the remedy. It should be remembered that spontaneous recovery is not impossible in primary cases.

APHASIA.

DEFINITION. Aphasia in its complete form denotes a condition in which the person is unable to communicate his ideas or wishes to others by spoken words, by writing, or by signs, in spite of the fact that his sight, hearing, and the muscles necessary for the expression of his desires are intact. There are many degrees and varieties of this loss, which have been specially named, with general acceptation, based upon the theory that definite classes of memories are stored up in definite cortical areas. Rather lately this has been challenged by Marie and Babinski, as will be stated later, but thus far with but partial acceptance.

In the first place, there are two grand divisions into Motor and Sensory Aphasias, according as emissive or receptive speech is primarily affected.

Motor Aphasia. If a person cannot speak at all, or only a few words, he suffers from Aphemia. If he has a difficulty in speech, in the selection, and possibly also in the formation of words to convey his meaning, he has Paraphasia, Confusion or Jargon Aphasia, or Stereotopy. If there is an inability to write he has Agraphia; while if, still being able to write, he writes wrong words, he has Paragraphia. An inability to sing is Amusia. An inability to indicate his meaning by gestures is Amimia. If he is unable to name objects he suffers from Anomia. These are all Motor Aphasias, and it has been suggested by Wilson (White and Jelliffe) that it would clear up our conception, if all motor aphasias should be considered several varieties of Apraxia. Apraxia is an inability or difficulty in performing the desired or accustomed act, while at the same time the object and its use are recognized, and the muscles are not paralyzed.

Sensory Aphasia. If the person is not able to understand words, sentences, or complex directions, he suffers from Verbal Aphasia, Mind Deafness, or Amnesia. If he cannot recognize or understand the use of things seen, touched, smelled or felt, he has Mind-blindness or Visual Apraxia. The inability to read is Alexia, Visual or Optic Aphasia, or Word-blindness. Wilson (as above) believes that it would be better to use the term Agnosia to cover all varieties of failure to properly interpret stimuli reaching the sensorium by any sense avenue whatever, though the stimuli themselves are appreciated by the corresponding cortical region adapted to receive such stimuli.

A Pure Aphasia is a condition where only one defect exists. It is rarely the case, but when it has been found it has been ascribed to the lesion of one of these specialized memory areas. Aphasias have been autopsied where the centers were found to be intact, but a definite subcortical lesion could be demonstrated. It was considered that in these cases the conduction tracts were injured, and therefore a second grand division seemed justified, and so aphasias were again differentiated into Cortical, and Subcortical or Conduction.

Differential Diagnosis. The only conditions from which Aphasias must be distinguished are Aphonia, Anarthria, Dysarthria, and Hysteria, Simulation, and Mutism as phases of a mental disorder.

Pathology. The function of speech is so dependent upon the perfection of the brain, that it is usually lost in any serious disturbance of that organ. Emotional shocks may produce such long-lasting spasm of the arteries that temporary aphasia results; we all are familiar with the effects of alcoholic intoxication, and other intoxicants may have a similar effect. When, however, the process consists in the formation of emboli in the meninges, or in the brain, or there is thrombosis or hemorrhage of the arteries, or fractures of the skull, a permanent condition of deterioration is inaugurated, and if it is upon the left side of the brain in a right-handed person, we often see aphasia following the paralysis. Along with the aphasia there are likely to be anesthesias, ataxia, asteriognosis, hemianopia, and hemi-deafness. It is a very common effect of thrombosis, since this is prone to affect the larger arteries, and a stoppage of circulation in the anterior cerebral will cause a motor loss, while if it is in the posterior there will be an auditory or visual aphasia. The double-sidedness of representation must always be considered. It is probably a fact that hearing, sight, smell, and taste are double in representation. Bastien claims that tactile and common sensibility are localized on the left side in the right-handed, and conversely in the left-handed. There is a great difference in the relative degree of loss in expression. It is a general truth that nervous impulse flows more readily along a well-used path, and therefore in an analogous way we very probably reason that words least used would be most liable to be lost, and the fact confirms the theory. Verbs, adjectives, adverbs and prepositions are used more than nouns, and very much oftener than proper nouns, and so proper nouns, nouns, and verbs are lost in about the order given. "Yes" and "No" are in constant use, and are rarely lost, and oaths often are retained, perhaps from constant use, or perhaps from their being more or less automatic, or a reflex act.

Physiology. It is certain that every convolution of the brain is connected with each of its neighbors with a system of fibres, then with the second from it, these two latter being also inter-related, and then with each of the other convolutions one by one, so that every convolution is in some sort of connection with every other one. If, now, it is granted that memories of all sorts are somewhere stored up, we can understand that such an individual is furnished with the material for speech. It has long been considered true that memories of sounds have been preserved in the middle portion of the cortex of the first and second temporal convolutions of the left half of the brain, in right-handed persons. This preference for the cortex of the left side in the right-handed persons pertains to all sorts of memories, and is inverted in the left-handed. When these sound memories are formulated into words, they are stored in the cortex of the marginal gyrus (which is that part of the descending parietal lobe that lies about the posterior extremity of the Sylvian fissure). We mentally appreciate the sight of objects in the cuneus, but memories of objects seen are retained in the angular gyrus (which is that part of the occipital lobe that lies about the posterior margin of the superior temporal fissure). Memories of muscular movements are stored in that part of the descending parietal lying just above the supra-marginal gyrus. The center for emissive speech (voice-production) is in the posterior portion of the third (lowest) frontal convolution, and the anterior portion of the insula (Broca's convolution), and the separated center for writing is in the posterior part of the second frontal.

Remembering now the elaborate systems of intercommunication between all parts of the brain, the process of education becomes a possibility. By the end of the first year of life, a child has begun to appreciate that there is a fundamental difference in the desirability of objects by which he is surrounded. He has found that persons sometimes gain possession of them by emitting certain sounds, and he struggles to gain a mastery of them. By the end of the second year he has appreciated them and practiced them to such an extent that he has rudimentary concepts, and a partial command of language. The amplification of these methods is all that is needed for the attainment of perfect speech. The tuition of the hand to copy forms, which he has discovered to possess the power to express the same concepts as the spoken words, completes the education necessary for the possession of emissive speech. A concept is an instrument of expression or appreciation, and it is made up by elaborating the bare suggestion projected into our consciousness by one of the mental associations, or indicated by some stimulus from the external world. It is developed into a complete, efficient concept by clothing the suggested object with every memory which we possess of its size, color, weight, smell, and all its remaining attributes. We are all familiar with this mechanism when we contemplate our mental processes on discovering an unfamiliar person whom we are desirous of meeting on terms of a half-remembered intimacy. We try to recall his dress on the former occasion, what he said, where he sat, in whose company he was, and perhaps what we ate. In fact, we go through this association process in every possible combination, and memory pictures of every description are hurriedly run over in order to reconstruct that formerly elaborate concept which we are now so anxious to possess. With variation in facility or difficulty, this is the history of the habilitation or rehabilitation of every concept. It is fundamentally varied from the fact that some people obtain their most distinct impressions from sight (visuals), while others appreciate more through the ears (auditives), but a far wider variation arises from differences in education, which is fundamentally a difference in the elaboration and sensitiveness of the association-tracts. It is axiomatic that frequency of use of the tracts makes conduction easier. The educated man appreciates far more, thinks far more, i. e., is more constantly re-assembling mental impressions into new combinations, and therefore keeps the tracts more sensitive and responsive. In the uneducated, there is a sort of inertia of conduction, and the growth of a concept is probably as laborious as their process of writing where we see the screwing up of the face, and the coincident movement of the tongue, while each word is repeated as it is written. The educated person runs the gamut of the memory centers with such rapidity that the process is without conscious effort, but probably is no different in kind with the learned than with the illiterate. The suggestion of a concept is appreciated in the anterior portion of the cortex of the frontal lobe. We invoke for its construction every variety of memory of its subject-matter, combining and rearranging associations, until it satisfies that co-ordinated activity of the whole brain which we call mind that the purpose has been accomplished, and then its emission is considered. The process will be inaugurated in the cuneus if we are visuals, and in the supra-marginal gyrus if we are auditives. Having, for instance, found the word in this gyrus, the conduction tract to Broca's convolution is stimulated, and we, as it might be said, speak the word to ourselves, at the same time energizing the tract to the superior temporal convolutions, listen to see if it sounds like our elaborated concept, and perhaps carry the picture to the cuneus, to see if it is the identical stimulus that called up the visual impression produced originally by the concept. Now, reassured by all these tests, the conduction tract to Broca's convolution is again stimulated, the cortical cells are discharged, and by way of a tract near the knee of the internal capsule (the aphasic fasciculus of Raymond and Artaud) an impulse is sent down to the proper nuclei in the medulla, and the muscles of voice production are set in motion. If written, the concept is sent to the posterior portion of the frontal convolution, where the finer movements of the hand and arm are represented.

If the Auditory centers (the middle portion of the two upper temporal convolutions, and the lower part of the descending parietal) are affected, there has been a lesion of the Sylvian artery, or one of its branches. If the type is pure the person can read writing and printing, but he cannot understand spoken speech, and has amnesic aphasia, or the sensory aphasia of Wernicke. He will be paraphasic, because he has lost the power of internally correcting his words before utterance, and tends to confusional and jargon speech. Unless he is a strong visual, he will be paragraphic. He will not be able to repeat from dictation, because he has lost sound-memories, but he can copy writing, because he still retains his sight, and his muscular memories. He, of course, cannot write from dictation, but he may be able to sing, since this may have become more or less a series of muscular memories. If the lesion is bilateral, he may lose, or have an impairment of memories for sound, and will have lasting word-deafness (amnesia), and in such cases his mind will show impairment. The lesion may be of any extent, and if it is very small there may be only a pure temporal lobe aphasia; or it may concern only the naming center, and then we have anomia. When the lesion is larger, and the lenticular zone is involved, there is added a greater difficulty in speech, which is jargon. There is also a hemiplegia, and we have a mixed temporal aphasia. Some sensory symptoms will usually be found.

Lesion of the Visual center, Occipital Aphasia, or pure Alexia. This is due to a lesion of the posterior cerebral, or a terminal branch of the Sylvian artery. This is characterized by word-blindness (alexia), and a agraphia (Dana says without agraphia), since he cannot remember the appearance of the written signs. For the same reason he cannot understand writing, but he can write from dictation, or may write spontaneously,.if it has become a mechanical act, and does not require interpretation from the visual center. If he learns to write with the left hand, he is liable to write from right to left, mirror writing, i. e., the words must be reflected into a mirror to be legible. This is not true of persons unaccustomed to write. The mental impairment is slight, and there is no hemiplegia, but if the lesion is not very superficial there is hemianopsia.

Lesion of the Motor Speech center, Frontocapsular Aphasia, or Aphemic or Agraphic Aphasia, Pure Motor Aphasia. This is due to a lesion of the ganglionic branch of the mid-cerebral artery. The person will be dumb (aphemic). He can understand spoken speech, and written partly. He cannot articulate correctly, and often is unable to write (agraphia). He cannot repeat from dictation, but can generally copy from writing, and he may not be able to make his wants known by signs (amimia), or his power in this direction may be limited. He has no word-blindness (alexia), nor mind-blindness (apraxia), nor mind-deafness (amnesia). Intelligence is generally preserved, but there is usually hemiplegia, without sensory symptoms.

Conduction Aphasia, Fronto-lenticular Aphasia, Mixed Aphasia. This is due to a lesion of the ganglionic branch of the mid-cerebral, usually the lenticulostriate. The simplest form is when the tracts running through the Island of Reil, and connecting the auditory and motor speech centers are severed. Aphonia is not always complete; ability to read and understand language will be impaired. Such persons can speak and write, but inasmuch as we continually correct ourselves by inward repetition of a word, and in this case the auditory center has been cut off, he may use wrong words, both in speaking (paraphasia), and in writing (paragraphia). This is only a probability, for while the connection between centers and Broca's is cut off, it is not from the ideational centers in the frontal. He can understand spoken and written speech, but in repeating from dictation he is paraphasic. If he is writing he will do it correctly, but if he is writing from dictation he is likely to be paragraphic.

This conception of aphasia, based upon an acceptance of Wernicke's localization of auditory, visual, olfactory, and motor memories in specific cortical centers, and Broca's definite centers for motor speech, has been attacked by Marie (Semaine médicale, May 23d and Oct. 17th, 1906), and his views have gained some converts in this country and abroad, from the fact that it had previously been noted that the post-mortem findings often had failed to conform to the clinical diagnosis. Marie bases his conclusions upon fifty autopsies in the Bicètre, in Paris. He asserts that Broca's assumption was based upon only two autopsies, that he had disproved Bernheim's case, and that Ladame had announced that the study of his case was not complete. Dejerine had in twenty-five years been able to find only one case of motor aphasia, due to a lesion in Broca's convolution, but based his belief on these cases of Bernheim and Ladame. Dercum quotes a case by Souques of a right hemiplegic and asphasic woman who was deficient in all modes of speech, especially aphemic and agraphic, who had been diagnosed as an aphasic of the Broca type. Three years later she died, and the lesion was found to be focus of softening, involving the posterior half of the two first temporal convolutions, the zone of Wernicke, and the third frontal convolution. Broca's center was intact. Fraenkel and Onuf (Zeitschrift für Nervenheilkunde, XV, Pg. 319, 1899) collected 104 cases of motor aphasia; seven showed a lesion limited to Broca's convolution. In five of these cases the aphasia was merely transient or temporary; in only two was it persistent. Marie denies that any such division as "motor and sensory" aphasia is tenable. He denies that memory centers exist, be they visual, auditory, or kinesthetic. He denies that the third frontal gyrus, Broca's convolution in part, has any functions connected with speech-mechanism. The speech-centers, according to his ideas are situated on the left parieto-temporal lobe, Wernicke's speech-center, which is a general region of intelligence, specialized for language, not a center for images as Wernicke thought. The classical motor aphasia of Broca is nothing but a general (sensory) aphasia; i. e., a language-intelligence defect plus a disturbance of speech (anarthria). The former is due to the implication of the Wernicke zone, the latter to an extension of the lesion involving the new hypothetical quadrilateral area around, and in the lenticular nucleus. The limits of the new quadrilateral area, which are very extensive and badly defined, according to the tract-anatomy, are anteriorly and posteriorly the vertical planes level with the anterior and posterior limiting sulci of the insula; bounded externally by the surface of the insula, and internally by the wall of the lateral ventricle. Above it reaches into the convexity; below it passes into the hypothalmic region. Such a revolutionary position has, of course, its opponents, as well as its advocates. Dercum (N. Y. Med. Jour., Jan. 5th, 1907), under Marie's inspiration, re-examined fourteen cases of aphasia, and attempted to divide them, as formerly, into motor or sensory aphasics. It was a failure, as it could only be said that they were all aphasics, among whom some showed a preponderance of motor speech disturbance, while others showed less of this symptom, or it was perhaps absent. All these cases showed intellectual deficit, which easily eluded discovery under the older methods of investigation, because the tests employed were too simple to develope it. As Marie pointed out, a word or two could be read, or repeated, or pronounced, but a series of two or three was given with difficulty, and the comprehension of five was impossible. He feels, however, that Marie's comprehensive claim that all aphasia is due to an involvement of the zone of Wernicke, and that all motor speech phenomena are due to an involvement of the zone which he lays out about the lenticular nucleus must be the subject of further research.

Campbell ("Localization of the Cerebral Function") agrees with Marie that a lesion of Broca's convolution cannot produce a lasting motor aphasia, but he does claim this upon other grounds. In the first place, the blood-supply is such that no lasting damage to such an extent could be caused by hemorrhage or thrombosis at this point; secondly, articulatory movements are not so closely circumscribed as that would demand. In his opinion, articulatory movements are represented in what he terms "the intermediate precentral area," of which the convolution of Broca is only a part. This larger area is devoted to the elaboration and co-ordination of movements produced by the precentral area proper. In the same way, the corresponding area in the second frontal performs the same function for the movements of the hand in writing. He bases his opinion upon his belief that the cells and cell-layers in this whole region are alike in character, and show no especial differentiation in the part termed the convolution of Broca. This is denied by Wilson (White and Jelliffe), who says that these centers for speech and writing are not those for the ordinary movements of these muscles, but have been found to be of an easily differentiated form, and situated slightly in the rear of the former cells. He thinks that the aphasic complex should be looked at from these two aspects: first, what is the pathological lesion; and secondly, what is the psychical and physiological disturbance resulting from it? Strictly speaking, the two aspects can never be divorced, but clinically we often see aphasias where no intellectual defect is apparent. Here Marie's caution that the tests must be complex might suggest a question. While he adheres to the classical division into motor and sensory, he would generalize the areas, as was suggested by Mettler in 1905, and drawing a line down through the fissure of Rolando, prolonged through the temporal lobes, would class aphasias from lesions posterior to this line as sensory, and anterior to it as motor. Moreover he would consider motor aphasia as an apraxia, which is an inability to perform certain movements with muscles which otherwise are not paralyzed. Broca's convolution may then be considered as an eupraxic center for the orderly coördination of successions of delicate muscular movements necessary for the production of spoken speech. In the posterior portion of the second frontal convolution is another eupraxic center for the production in orderly sequence of the finer movements of the hand and fingers in writing words. On the sensory side, when a patient is unable to recognize what he sees and hears, he suffers from agnosia, which corresponds to apraxia on the motor side. This term covers all such expressions at mind-blindness, object-blindness, tactile aphasia, optic aphasia, etc. Where the centers of Broca and Wernicke are cut off from the ideational centers, we get what is termed transcortical, or conduction aphasias, the pathology of which does not rest upon a very well-established foundation.

Dana (New York Medical Journal, Aug. 10th, 1907) agrees with Marie that a division of aphasia into motor and sensory is not tenable. He also agrees that an intellectual deficit is generally an essential factor, but he does not agree that Broca's convolution has nothing to do with motor speech, nor does he believe that this defect comes from a lesion in the zone of the lenticular nucleus, although the fibres necessary for the production of speech pass through some part of this nucleus. He seems rather to agree with the conclusions of Campbell, when he says that a complete aphasia from a cortical lesion here is impossible, on account of the peculiarity of the arterial supply. The complete aphasia which does come from lesions in this neighborhood is due to the fact that the lesion destroys the converging fibres from, or associating fibres to this convolution. As these pass over, around, and to the inner side of the lenticular nucleus, the statement of Marie that complete anarthria, or pure aphemia is caused by lesions here is correct, but the aphemia is due to the cutting off of the impulses from the third frontal and temporal convolutions, and not to a destruction of peripheral speech-centers in or about the lenticular nucleus, for there are none there. We have then the psychic area, or the zone of language in the frontal, temporal, and parieto-occipital lobes, and aphasia occurs in different types, according to the area affected. According to his theory, he would make a fronto-capsular lesion an aphemia, and covering what is usually known as pure subcortical motor-aphasia. This is a rare form. These patients have a very marked right hemiplegia, with no sensory symptoms. The person cannot speak a word, nor repeat, nor count, but he can write, read, understand, and he has good general intelligence. Slight variants from this pure type show ability to swear, and repeat words (ejaculatory language), and have some involvement of writing and reading. Such cases can only be caused by a frontal lobe lesion extending into the lenticular zone on its inner side, not involving the upper part of this zone, or the superior longitudinal fasciculus. They are fronto-lenticular lesions, and must be due to a lesion of one of the ganglionic arteries, probably the lenticulo-optic, which does not supply much of the corpus striatum. When, as in Bramwell's case, it affects the outer and upper part of the lenticular zone, it destroys the superior longitudinal and arcuate bundles, and causes some symptoms of sensory aphasia as well as aphemia.

Occipital or Parieto-occipital Aphasia (Pure Alexia). The occipital lobe is supplied by the posterior cerebral artery, and when lesions occur in it, they involve the intermediate visuopsychic centers, or the tracts connecting it (inferior longitudinal bundle) concerned in reading. These are situated in the angular and second occipital gyri. When this occurs the patient can talk well, and understand, and can write well, but he cannot read, except a few words or letters, and naturally he cannot copy well. He always has hemianopsia, and the alexia is sometimes wrongly attributed to this. There is sometimes hemiataxia, or anesthesia, or hemi-paresthesia, which may be temporary. The intelligence is good. These cases are rare. Alexia may be caused by lesions of the Sylvian artery also, since it supplies the angular gyrus, but generally in such a case we get some injury of the temporal cortex, and with such a lesion we find anomia and mind-deafness.

Temporal, or Parieto-temporal Aphasia (Sensory Aphasia of Wernicke). The artery of Sylvius in its terminal branches supplies the first, and part of the second temporal convolutions, and the inferior parietal, including the supramarginal and angular gyri. The essential language center is then affected, and we should expect word and mind-deafness as the dominant symptom; but this is not the case, or is so only partially. Anomia and paraphasia, without much or any hemiplegia, are the most striking symptoms. The patient has some voluntary and responsive speech, and talks for a time rather fluently, but having loss of power to name objects seen, or to use verbs and sentences properly, he is paraphasic, and after a time becomes confused, and may speak only jargon. He can read but poorly, and while he can write and copy a little he does it very badly. He can sometimes understand questions, and simple directions, but cannot comprehend complicated statements, and at times is childish and emotional. The physical symptoms vary, there may be a temporary hemiplegia, usually some hemianesthesia, or ataxia, or asteriognosis, but this does not always last. Hemianopsia of variable duration may be a symptom. In these cases there must be some degree of cutting off of the association tracts between the auditory psychic area, and the visual area. The inability to read, and write, and copy seems to result from the fact that the most important of the areas concerned with the function of language, and the more recently acquired parts of that function, viz., that of reading and writing, would naturally go when the machine is badly injured. There are two varieties of this aphasia. When the Sylvian artery is involved nearer its origin, the lenticular zone is encroached upon, and we have a tempero-lenticular lesion. The patient has a defect of intelligence, with decided hemiplegia, and an absence of sensory symptoms. There is a great defect in voluntary speech, a violent and nonsensical use of certain words and sentences, a violent jargon, or stereotopy. The patient understands poorly, he can read a little, and can write a little. The dominant symptoms are: jargon, stereotopy, dyslexia, dysgraphia, some mind-deafness, hemiplegia. The second variant is when a small branch of the Sylvian artery supplying the temporal lobe is involved. There is a pure anomia or optic aphasia. The patient can talk well, reads, writes, understands, but he cannot name objects seen, and often he cannot name objects felt, heard, smelled, or tasted.

Fronto-lenticular, or Lenticular Zone Aphasia (Mixed Aphasia). Most aphasias are of this type, which comes from a lesion of a ganglionic branch of the middle cerebral, usually the lenticulo-striate artery. This supplies the corpus striatum, internal capsule, and the fibres converging to it from the second and third frontal (anterior part of the internal capsule), and from the precentral convolution. With such a lesion we get aphemia, which is generally very complete, some alexia, and some difficulty in understanding language. There is dyslexia perhaps, instead of alexia, agraphia or dysgraphia, some word-deafness, and some impairment of intelligence. The physical losses are hemiplegia with no sensory symptoms, and it is the condition termed by Marie as anarthria plus aphasia of Wernicke. Dana thinks that the use of the term anarthria is unfortunate, since these patients can speak a few words, and those plainly. They cannot talk, because of the loss of articulatory memories, and also probably because of the severe injury to the association tracts from the tempero-occipital cortex (superior and inferior longitudinal fasciculi). This causes their difficulty in reading and understanding language.

Diagnosis. (Based upon Beevor's scheme.) We should first inform ourselves whether the person is right or left-handed, whether sight and hearing is intact, or to what degree it is impaired, and whether there is a defect in the muscles of phonation or the speech apparatus. We should also inform ourselves as to the education and station in life of the patient before estimating the degree of mental defect shown by our examination. Discover whether the patient can spontaneously utter intelligible words, whether many or few. If he talks fluently, see if he misplaces words or syllables, or speaks in disjointed phrases, or in jargon. Does he understand words which he hears? This may be tested by asking him to touch his nose, eye, and ear in turn, and it also shows his comprehension of nouns. If we then ask him to smile, whistle, or shut his eyes, his success will reveal his command of verbs. Ask him to define beauty, sorrow, or darkness, and we gain a knowledge of his comprehension of abstract ideas. A patient may execute one command correctly, but simply repeat the first action as an answer to subsequent and different ones. A patient may be asked to hum a tune; he may be able to sing, while he is unable to repeat the same words in a speaking voice. See if he can write spontaneously, if it is intelligible, or if words and syllables are misplaced, or if it runs off into jargon. If the right hand be paralyzed, let him try with the left, but the result may be mirror-writing. Test him with written questions, and commands which he sees; write down and show him sentences of a simple character. Find if he can copy from printed letters to written. See if he can write dictation, i. e., words which he hears. Place in front of him a number of objects in common use. Name them one by one, and see if he can pick them out. Ask him to repeat words pronounced to him. See if he can name objects seen, and if he can read aloud from words shown him. Point to different objects, and ask him what they are. See if he understands gestures, and pantomimic movements. Without speaking to him ask him to imitate you when you perform various simple acts, like touching the nose, ear, etc. Give him various articles in common use, and observe his recognition of the various criteria of weight, shape, color, size, taste, smell, and the like, and also if their purpose is recognized, and if they are applied to their proper use.

DIFFERENTIAL DIAGNOSIS. Aphonia is the condition where all language is perfectly comprehended, but speech is impossible because the vocal cords are paralyzed, as will be evident upon laryngoscopic examination.

Anarthria is a condition where tests will show that intellect and comprehension of all forms of expression are perfect, but there is a total paralysis of the vocal apparatus.

Dysarthria is a similar condition as to the comprehension of speech, but language is poorly enunciated, varying according to the different degree of involvement of particular sets of muscles. Bulbar speech is an example of dysarthria.

Mutism may be a symptom of mental disease, and no diagnosis of aphasia should be made until one is certain that it is not a case of mental derangement. The mental reduction spoken of by Marie as the basis of all aphasia is by no means a systematized derangement of the mind, but rather a slight reduction along certain lines.

PROGNOSIS. Time alone can tell whether the aphasia from a shock to the brain is functional and temporary, or organic, and in some sense permanent. Very slight causes have produced temporary aphasia, and while an injury to the brain may have been severe, it must be kept in mind that the aphasia may not be a result of the injury which holds our attention, but may be due to some collateral, temporary, or removable condition. Such conditions cannot be accurately estimated in less than a week. Word-deafness has a better prognosis than motor aphasia. Much depends on age, since in a child, or young adult in whom speech has become established, the re-education, or substitution of an intact right center for a damaged left center is much more probable than in an older person. Before the age of seven, both hemispheres are used indifferently, but after that age it is definitely localized in the left-hemisphere in right-handed persons. Investigation should be made for prognostic purposes, into whether the person is naturally right-handed. If naturally left-handed, the speech center may be found on the right side, and so have escaped injury. In a general way it may be said that the prognosis is worse by the amount of delay in showing improvement; the later the stage of life, since less arterial compensation can be expected; the greater the severity of the causative lesion; worse in hemorrhage than in thrombosis, because in the first instance we are dealing with destruction of nervous tissue, while in the latter there may be a removable ischemia; worse when definite arterial changes can be demonstrated. Where speech has never been established, although there was no mental defect, no rule can be given, but some remarkable recoveries have followed the educational methods employed in teaching the deaf. In other cases where speech had been established, recovery has been known to follow mental or physical shock; here also educational methods have been employed with success. If a person is a strong visual, his recovery from agraphia is more probable than if he were an auditive.

TREATMENT. Re-education is the remedy, and often touch is better than the presentation of the object to sight alone. Sometimes the vibrations of the larynx of the instructor, as in teaching the deaf and dumb, are the only possible means of awakening the impaired faculties.

CHAPTER XII.

TUMOR OF THE BRAIN.

DEFINITION. This is the formation of new growth in the brain, and the term is elastically enlarged to include growths in the cranial cavity which compress or irritate the brain, without actually being developed on, or within it. All the common forms of tumor are possibly located here, and some are peculiar to this locality.

SYMPTOMS OF A TYPICAL CASE are headache, double choked disc, vomiting and vertigo, with retardation of the pulse, localized spasm, especially apt to be followed by a persistent paresis or paresthesia, somnolence, and mental dullness.

DIFFERENTIAL DIAGNOSIS: This is from Chronic Nephritis, or Contracted Kidney, Profound Anemia in young girls, Chronic Lead Poisoning, Tabes, Hydrocephalus, Chronic Cerebritis or Encephalitis, Acute Encephalitis, Meningitis, Pseudo-Tumor, Abscess, Cerebral Hemorrhage or Softening, General Paresis, Epilepsy, Migraine, Disseminated Sclerosis, Hysteria, Intracranial Aneuryism, Alcoholism, and Multiple Neuritis.

AGE. It occurs in persons at all ages, one series of 70 cases (Brush), showing a range of from the sixth month to the seventieth year. Ballance removed a large tumor from the cerebellum of a girl of four years and ten months, and Hepfner, from a girl of six years, took a cerebellar sarcoma which occupied almost the whole of that structure. The greatest frequency of tumor is in the fourth and fifth decades of life, but definite differences characterize the growths, which will probably be present at the different periods of life. Tubercle is most probable in childhood, and decreases in frequency as the years go by, until it becomes highly improbable after the thirtieth year although it has been found at the sixtieth.

Parasitic tumors are found in childhood and early life. Sarcoma, glioma, syphiloma, and endothelioma occur at any age, but are the tumors usually found in early middle life, while in late life glioma is not found, but cancer is found instead. However, tumors of any kind are rare after 60.

SEX. Males are more often the victims than females, and this may depend upon the etiology. In 70 cases as above 41 were male, and 29 female.

ETIOLOGY. This is often a matter of conjecture. Cicatrices can often be found at the site of origin of a tumor, but they must be supposed to have a previous injury as their determining cause. Injury, however remote, has been considered a competent cause of tumor, but it is now generally considered that most often traumatism accelerates the growth of an existing tumor, rather than originates the new growth. They sometimes arise from anomalies of developement, and gliomata are most frequently the result. Infections, metastases, and also parasites, as actinomyces, hyatid, and cysticercus, have caused tumors.

PATHOLOGY. There seems to be a difference of opinion concerning the relative frequency of the various varieties of tumor. Tooth analyzing 500 tumors says that 49% were glioma, while Starr from 300 cases finds that tubercle is most frequent, but it is certain that whatever may be the individual order, the majority of cranial tumors are either Glioma, Tubercle, or Sarcoma. Histologically, there is undoubtedly a distinction between innocent and malignant growths within the cranium, and there is no doubt that the sarcomata tend to grow more readily, but on the whole the central nervous system does not seem to furnish very favorable tissue for the growth of malignant tumors. Although it may be urged that an intracranial sarcoma kills by compression before it has had time to disseminate, yet in cases of widespread secondary deposits of malignant disease, occurring in many other organs, it is rare to find any in the central nervous system, and in cases in which cerebral sarcoma has been removed, general metastatic recurrence is unusual. (Hall.)

Glioma. Gliomata are a product of the neuroglia, are almost always single, and are always primary, except when an extension fom glioma of the retina. They vary in size from that of a hazelnut to that of one's fist, and are yellowish-white to a reddish-gray in color. They often closely resemble in consistency the normal brain-substance. They are softer than a sarcoma, are very vascular, and if hemorrhages have occurred, and such hemorrhages are of frequent occurrence, they may be taken for spots of softening. They may resemble a local hypertrophy from the similarity of their color and consistency to the normal brain.

The glioma, when it enters upon an active stage, by the very exuberance of its activity carries on within itself a process of self-immolation. The blood-vessels which may increase vastly in numbers (an angiomatous stage) become blocked (intimal proliferation stage); thrombosis spreads backward into the larger vessels (a thrombotic stage), and lastly there results necrosis of the area concerned. The hemorrhagic tendency makes them liable to contain cysts, and it is held by some that all cysts in the brain, which are not the results of hemorrhage, or softening, are features of a diffuse glioma. *Cavities lined by cells* are, however, rarely gliomatous. It is advisable to remove part of the cyst wall to assure oneself that it is a cyst, and not a cystic degeneration of a glioma. In breaking down they may undergo a sarcomatous degeneration, which is probably the origin of Glio-sarcoma. They extend by infiltration, and not by compression, and their rate of growth is slow, often coming to a standstill, and if rapid is almost always the result of a hemorrhage. They are never capsulated, and so cannot be successfully removed, and an attempt to do so stimulates their growth. Their especial localization is in the hemisphere, or the cerebellum, more in the white than in the gray matter, and they are rare elsewhere. They do not involve the meninges or the bones.

Sarcoma. This differs from glioma in being a connective tissue tumor, and tends to develope from the periphery, and injure the brain rather by compression than by infiltration. It arises from the meninges, the periosteum, and the bones of the base of the skull (temporal, sphenoidal, and less often the occipital), and occasionally from the connective tissue about the blood-vessels. This peripheral origin makes the cortex and the cerebellum the apparent point of origin of sarcomata. They are practically always solitary, since the multiple form of sarcoma, melanotic nodules, is almost unknown in the brain. There is, however, a diffuse form at the base of the skull which involves all the structures of that locality. They are harder than glioma, are not vascular, and possess a capsule, or lie in a zone of softened brain tissue, and therefore present a better prognosis for removal than the other forms of brain tumor. They may be metastatic, in some cases are the site of hemorrhage, and they degenerate and caseate; this is especially the case in the tumors of the cerebellum. Under such conditions they are difficult to differentiate from a glioma, and are the form known as glio-sarcoma.

Tubercle. Its point of origin is from an infected focus in the lymphatic sheaths of the blood-vessels. It is multiple as a rule, and when so, tends to symmetry of arrangement. (Oppenheim.) It is practically always metastatic. Solitary tubercle is also found (20%), and is similar to a gumma, both having a poor blood supply, and tending to caseate. The tumor is usually accompanied by a miliary tuberculosis of the meninges, which if discovered is diagnostic. The tumors are nodular and rounded, and while generally like small nuts in size, may be as large as the fist. The growth is typically rapid, but at times is slow, and there may be periods of stasis, and even of regression. The peripheral portions have the characteristics of granulation tissue. No portion of the brain is immune to the invasion of tubercle; in children it occurs most often in the cerebellum, while in adults it is found equally in the pons, cerebellum, and the cerebral cortex. Like a sarcoma, it does not infiltrate, but compresses. On account of its similarity to the other neoplasms, the only positive diagnostic feature is the discovery of the tubercle bacillus in the tissues of the affected person, but it may be strongly suspected if there is an hereditary tendency in the individual to tuberculosis, if he has been exposed to conditions favoring its developement, and if there is a history of typical symptoms of the disease, and a primary, pulmonary, intestinal, joint, or glandular infection. The tuberculin test should always be employed.

Syphiloma or Gummata. These are just the opposite of tubercle, in that they are rare in childhood; they are never the result of hereditary syphilis. They may appear at any time after infection, from one up to twenty years. No matter where it appears, it almost always arises from the meninges, and a connection can be traced between it and the pia. The other point of origin is the wall of the vessels. Unlike sarcoma, multiplicity is the rule, but like sarcoma and tubercle, it is non-vascular and liable to caseate; as a rule it does not grow to as large size as tubercle. The typical developement of gumma is in the form of a clear gelatinous coating of variable thickness on the membranes of the base of the brain, where it mats all the structures together and destroys them, functionally at least, by its subsequent contraction. Sudden increase in symptoms, and as sudden a regression, is quite diagnostic, but the Wassermann test has given certainty instead of tenable hypothesis, and so also has the possibility of discovering lymphocytes in the cerebro-spinal fluid. Alone among cerebral tumors it is amenable to medication, for antisyphilitic medication will at times clear up the most desperate cases in a few months.

Carcinoma. This is that variety of malignant growth which is nearly always metastatic, when found in children, from growths of the orbit. It is, however, a rare tumor at any age, patients are almost always over fifty years of age, and it is then subsequent to prior growths in the orbit, retina, or breast. It may arise from the dura or any other of the structures of the brain. It is soft, with irregular outline, and very vascular. Its contents are colloidal, and it softens into a pultaceous mass. It is one of the large tumors, and may reach the size of a child's head; it is ordinarily multiple, of rapid growth, and is rarely encapsulated. It may be circumscribed or diffuse, and rather affects the central ganglia as a site, and acts both by compression and infiltration. It is the least susceptible of successful operation.

Psammomata. When sarcomata develope from the dura, and are slow in growth, they may have in them calcareous deposits, and then are called Psammomata.

Cholesteomata, angiomata, and dermoid and parasitic cysts are seen at times in Europe, but are infrequent in this country. As they act only by compression, the symptoms are apt to be doubtful, and they may give rise to no symptoms. If they can be localized, they are removable, as they possess a capsule. They are often multiple.

Chloroma is a recently discovered tumor, flat and greenish, affecting generally the bones of the skull, especially those of the orbit and base of the brain, but occasionally appearing in various lymphatic glands. Chloroma is a primary disease of the hematopoietic (the blood-making) organs, with special tendency to the formation of malignant growths of green color, in various localities, but chiefly at the base of the brain. (Starr.)

Chordoma is a small tumor found occasionally in the median line of the clivus, near the union of the sphenoid and occipital bones. It is colloidal, and is the size of a small cherry.

LOCALIZATION. Over one-third of all brain tumors are in the posterior fossa; about one-fourth are in the cerebellum, or cerebello-pontile angle, and about an equal number in the cortex cerebri. Very few tumors are found in the medulla, or fourth ventricle.

About one-twelfth of all tumors are in the basal ganglia and lateral ventricles, and a rather smaller number in the mid-brain (corpora quadrigemina and peduncles). The order of frequency in location, then, is about as follows: cerebellum-pons; cortex cerebri; centrum ovale; pons; mid-brain; medulla.

In children their relations are a little different. Here nearly one-half the tumors are in the cerebellum and pons. They are very much less frequent in the cortex cerebri, but more common in the centrum ovale, basal ganglia, and mid-brain, so that the order of frequency is: cerebellum-pons; cerebrum ovale; basil ganglia; mid-brain.

As the mid-brain as well as all the contents of the posterior fossa are nourished by the vertebral arteries and their branches, it follows that the majority of tumors in children are in this vascular area. A little over one-eighth of the total brain mass is thus especially subject to tumor growth. (Dana.)

CHANGES RESULTING FROM THE PRESENCE OF TUMOR. In the description of the various kinds of tumors, it will be noticed that while glioma, tubercle, and carcinoma infiltrate the brain-tissue, sarcoma, syphiloma, and the unimportant varieties of tumor and cyst usually injure the brain only by compression. Some produce both effects, while others are too small to produce much pressure. Most symptoms are produced by compression. By this the convolutions are flattened, distorted and pushed from their places, and the pia and the brain generally are dry and anemic. The brain is softened, where a growth impinges upon it. A growth can scarcely be so small as not to interfere with circulation, and a wide range of symptoms is thus entailed. The cerebro-spinal fluid is increased, and hydrocephalus is a common accident, particularly in case the tumor is near, or in the cerebellum, since the flow from the great Galenic veins is impeded. Infiltration of the sheaths of the emergent nerves takes place, with resulting changes in them, and they are also flattened by the pressure. This is most marked in the abducens and the olfactory. The spinal cord may be the site of changes, both from pressure and interference with circulation. Pressure, both local and general, may thin out, or even perforate the skull, and the sutures are usually separated in young children.

SYMPTOMATOLOGY. Surgical technique has been elaborated to such a point that at present a more hopeful prognosis can be given to operations for the removal of new growths of the central nervous system, but early diagnosis is an almost absolute pre-requisite for success. The recognition of the early symptoms of tumor, therefore, is of paramount interest. Slight difficulties of coördination in movement, slight changes in muscular tone, as, for instance, a slight loss of knowledge of attitude, or muscular sense in one hand, slight disabilities, or perversions of the special senses, should always excite suspicion. Horseley says that atopognosis (Monk), or loss of memory of motions may be present long before any other motor loss, while asteriognosis does not occur until the motor paralysis is well established. Convulsions are an early symptom, and if focal can hardly be due to anything but local irritation. Karpas quotes a case where they antedated all other symptoms by three years. The fundus oculi shows early changes in 70-80% of the cases. Cushing says that the earliest sign, even with little or no changes in the fundus, is an interlacing of the color-fields. While all colors may be affected, it will be most evident in the blue or red grounds. Mental dulness, emotionalism, or other changes in character, aphasia, or amnesia should excite our suspicions.

The symptoms are General and Focal. The General symptoms inform us only that there is a tumor in the cranium, while Focal symptoms, when capable of interpretation, point to the particular portion of the brain affected.

GENERAL SYMPTOMS. Headache is an almost certain and constant symptom, and is generally the initial one. It is not always initial, or constant, but recurrent, and few patients escape it. It is usually diffuse, and not of particular localizing value, but is more apt to be severe if the growth is under the tentorium. It is produced by a stretching of the sensitive dura, and the branches of the 4th, 5th, and 12th cranial nerves, and the sympathetic fibres which innervate it. The pain is most constant and severe, especially at night, in syphilitic growths.

It is apt to cause a discernible sensitiveness of the scalp over the site of the tumor, and the cranium is more tender to percussion in that area, and if accompanied by a bruit it may localize a tumor in the cortex, but it should be remembered that chronic lead poisoning may produce as severe a grade of pain. If very severe, and extending down the back, it suggests a growth in the posterior fossa, or in the cerebellum. Children suffer less from it, on account of the ease with which separation of the sutures is produced. It is aggravated by anything which suddenly increases the pressure within the skull, like coughing, sneezing, or sudden changes of position.

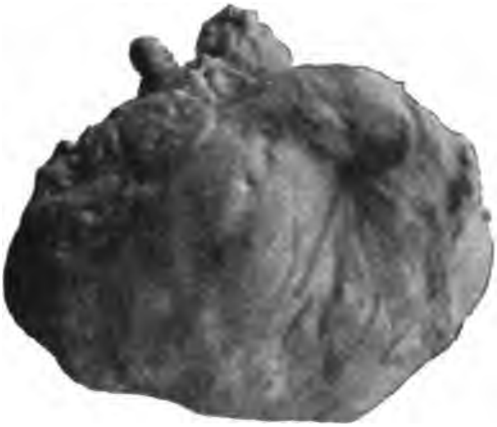


FIGURE 83. CEREBRAL ANGIOLITHIC TUMOR. (Actual size.)

Ocular Symptoms. Choked Disc. This is the condition where the head of the optic nerve becomes congested, and opaque, and loses its clear outline. Its typical termination is in optic atrophy, and loss of sight, but this is not always accomplished, and loss of sight may be such a late result that the ophthalmoscope is a necessity for its recognition. In children we are more apt to find loss of vision as a primary symptom. Choked disc may antedate the headache. Primary optic atrophy is never found in tumor. It is always secondary, and indicates a tumor of long standing. Choked disc occurs in from 80 to 90% of the cases, and is almost always bilateral, although it is often more marked in one eye than in the other. It may be present in a high degree without impairment of vision, and is always present in tumors of the corpora quadrigemina, in 89% of tumors of the cerebellum, and absent in two-thirds of the tumors of the pons, medulla, and corpora callosum. It is least frequent in tuberculous, and most common in glioma and cystic tumors. In the rare cases where it is unilateral, it is ascribed to the location of the tumor in one nerve, or in front of the chiasm. It is produced, probably, by an inflammation resulting from the forcing of the cerebro-spinal fluid into the sheaths of the nerves. This sets up an interstitial inflammation which later affects the nerve fibres, but initially stops the venous return. It may result from the action of toxic products released by a primary inflammation of the disc. Cushing, Jour. A. M. A., July, 1911, says that a slight turning in of the blue field at the periphery is all that we can expect to find in a cerebellar tumor. He quotes a case of Jacksonian epilepsy, who had no pressure symptoms, and only a slight contraction of the visual fields, but marked interlacing, and on operation a benign tumor as large as one's fist was found. This symptom may be due to pressure. Some other conditions may show it, viz., abscess, serous meningitis, etc. It may affect all colors, but when blue and red have been examined, about all has been done that it is best to do with such feeble patients. In ten out of fifty-three cases the dyschromatopsia preceded all other changes in the eye-grounds, or accompanied the most primary tendencies toward choked disc. Generally the greatest interlacing is found on the same side as the tumor. Transient attacks of dim vision or blindness frequently recurring, and lasting from a few seconds to half an hour, constitute a very important symptom of cerebral, most frequently cerebellar tumors. It may be due to interference with the blood supply of the occipital lobe, to temporary strangulation of the nerve, to increased intracranial pressure or to pressure upon the chiasma by the infundibulum. (Wm. Spencer, Hahn. Mon., Oct., 1906.)

Mental Changes. The mind usually shows the effects of cerebral pressure in a change of disposition, generally a mental dulling, but sometimes in irritability. The patient may be melancholy, and seek solitude, or he may be delirious and excited. The cause may be compression, but in the absence of any general symptoms, the patient may display a peculiar mental weakness with exaltation. It might be ascribed to the causes producing headache, but has been seen in its absence. Some change is almost always present. I saw a case, however, in which, until a late period, there was undisturbed mental ability, and unimpaired equilibrium. Mental changes are more apt to occur in large tumors, and those of the frontal lobe.

Stupor. A stuporous condition is very characteristic. It may be present at all stages, and the patient will first appear sleepy and apathetic. He will next actually fall asleep, if not especially interested; later he will fall asleep while talking or eating (narcolepsy), which is said to be especially true of tumors of the corpus callosum, and in the last stages there is a condition of lethargy approaching coma. The patient can be aroused with difficulty, and is apt to defecate and urinate unconsciously. This symptom may be absent if the tumor is very small, or if it lies outside the brain.

Insomnia. Insomnia is apt to be a symptom at some stage of a case, since it is the result of irritative and inflammatory conditions, which are extremely liable to be part of the pathological changes. It is the opposite of somnolence, which is a compression symptom. The insomnia may also be the result of the intensity of the general symptoms. It is more often found in adult cases than in those of children, and most frequent and pronounced in those arising from syphilis.

Vomiting. This is not always seen, but is rather dependent upon the rapidity of the growth of the tumor, or upon the fact that it is localized in the cerebellum or pons. It may be a symptom of tumor in any part of the brain. It does not become prominent until headache has become a factor, and is often an accompaniment of its acute exacerbations. It is most common in children. It is cerebral in type, in that it is often unaccompanied by nausea, does not depend upon the character or quantity of food, is very sudden and violent, and accompanied by great prostration, and is projectile. Change of position may bring it on, and it may be so continuous as to threaten life by interference with nutrition.

Vertigo. This is a symptom of irregular appearance, and is quite dedependent upon the location of the growth. It is peculiarly marked in cerebellar tumors, and often with those of the pons, and structures in the posterior fossa, because they compromise the functions, directly or indirectly, of the auditory nerve. While actual vertigo, where the patient reels and must grasp objects for support,. is uncommon, he is very commonly subject to an unsteadiness like the results of moderate intoxication. Equilibration is so complex a process that localization of a disturbing cause is very difficult. These patients may escape vertigo, or it may be an intermittent symptom, but they all complain of a feeling of pressure, pulsation and discomfort in the head. They may have a vertiginous condition from implication of the motor oculi, auditory from implication of the 8th, and forced movements and attitudes from tumors in the middle lobe of the cerebellum.

Irritation of the dura is almost a necessity of the existence of a brain tumor. The nerve supply of the dura is mainly from the fifth nerve. A powerful peripheral stimulation of a nerve irritates its nucleus, and on account of the inter-relation of all of the nuclei is liable to injuriously affect the more sensitive ones. In this case it is probable that a secondary irritation of the vestibular and pneumogastric nerves is the cause of the vertigo and vomiting as well.

Convulsions are common. They may be like epilepsy, of either the grand or petit mal form, or they may be Jacksonian at first without loss of consciousness, but consciousness is lost when the convulsions become general.

Circulatory and Respiratory Disturbances. Fever may occur from inflammatory changes in the brain or meninges, and it has been said that local elevation of temperature can be discovered on the scalp over the growth. This is questionable, but possible if the growth is cortical.

The pulse is slowed (compression symptom), as low as from fifty down to thirty beats per minute. It is apt to be irregular. A fatal termination, or a direct action upon the vagus, is indicated by rapidity of pulse rate after a preliminary retardation.

Respiration is usually affected in some manner. It is apt to be slower than is normal, and subject to periods of irregularity. It may be Cheyne-Stokes in character at times, and then resume a more normal rhythm, but that condition usually presages a fatal termination.

FOCAL SYMPTOMS. These are the symptoms which are sought for to enable us to localize the lesion which the general symptoms have led us to think to be tumor. These are of two varieties, direct, and indirect. The anatomy of the brain must be fully grasped in order that we may appreciate that one symptom can be due to the direct pressure of the growth, while another is manifested by the disturbance of a distant center, from the fact that the growth which caused the first symptom directly, has also caused the second by pressure upon the motor tract leading to the distant center referred to, and to which the motor or sensory disturbance must be referred. It is then a question which is the direct, and which the indirect symptom. This is decided by the order of appearance of symptoms, and also from the fact that no direct focal injury is without associated symptoms of a disturbance of contiguous areas. Slight pressure symptoms indicate a small tumor, and that the focal symptoms are direct. Tumors are first to be localized in the motor cortex, or at the base, before a finer localization is attempted. If at the base, there will be some degree of implication of the cranial nerves.

Focal symptoms are few, and they will always be the same for a tumor in identical localities, and they will be unilateral if the tumor is single; but multiple tumor is not unknown. They are unilateral, since no tumor is near enough the middle line to produce bilateral symptoms, and tumors even of the corpus callosum rarely produce bilateral effects. Tumors may attain large size, and produce no focal symptoms, if they grow slowly, and act by compression rather than by infiltration.

Frontal Lobe. Such a localization is probable if we have pronounced general symptoms, and an absence of focal ones. Changes in mentality are not focal symptoms, since this function is probably diffused over the whole brain, but it is considered that *early* mental loss, the appearance of a mild dementia without delusions, changes of disposition, personal uncleanliness, and a loss of the sense of decency should influence the diagnostician in localizing the growth in the left frontal, since such losses appear *late* in tumors of other localities. This has been marked in tumors of the frontal cortex, and underlying white matter, but is not found in tumors at the base of the anterior fossa. The only definite symptom of a tumor here is an aphasia of gradual onset, and slightly pronounced character, and a reluctance to speak. This localizes the growth in the cortex of the left third frontal in right-handed persons, and in the right in left-handed. A tumor in the frontal cortex may produce motor spasms and paresthesias by a propagation of impulses to the rest of the brain, and there may be a disturbance of equilibrium, since here are located cells whose fibres go to the cerebellum of the opposite side. If the tumor is in the lower surface of the lobe, it will compress the optic and olfactory nerves, and will interfere with the sense of smell, and may produce hemianopsia, and a severe form of optic neuritis. If it invades the orbit, the globe will protrude and be paralyzed. Conjugate deviation of the eyes with muscular spasm in the arms or face make very positive a diagnosis of a tumor of the frontal lobe. The eye symptoms with rigidity of the neck and back imply the same condition.

Langdon and Kramer (J. A. M. A., Dec. 3rd, 1910) report an encapsulated, nodulated, endothelioma arising from the pia mater, involving the middle third of the left precentral convolution, growing diagonally backward across the Rolandic fissure, displacing but not invading the postero-central structures. It was therefore in the psychomotor area, and gave rise to visual hallucinations, somatic delusions, astereognosis, and right hemiplegia, but headache, vomiting, vertigo, and papillitis were absent, although it weighed 26 grams.

Tumors of the Temporal Lobes. The right temporal, in right-handed people, is considered to be a latent region, and a tumor will produce general symptoms only as a direct result, but by its pressure upon the brain stem and neighboring parts it may produce many confusing symptoms.

Moisset-Bentler report a glioma of the inferior portion of the tempero-sphenoidal with psychic symptoms only. If the pressure is backward there may be optic aphasia, while if it is downward there may be vertigo with forced movements, and a mixed type of aphasia. We may also see hemianesthesia, hemiplegia, and hemianopsia, and also ataxia. If it is on the left side in a right-handed person, it may injure the uncinate gyrus (same as hippocampal, and lies inward from the superior temporal) and directly produce loss of taste and smell, while if it affects the first and second temporal gyri there may be word-deafness, and the person affected will be unable to recall the names of persons and objects, and lose his power to understand spoken language. This constitutes amnesic aphasia.

but while deafness might theoretically be supposed to occur, it is a very rare result, since audition is probably represented in both hemispheres. Auditory hallucinations may, however, result. In one case there was an aura of fear referred more especially to the epigastric region, which spread upward, and then forward, producing first a sensory, and later a motor focal epilepsy by means of irritation of the post-central, and precentral gyri. In the meantime the stimulus spread in a forward direction, and there produced a true uncinate spasm, a subjective sensation of a foul odor like rotten weeds. Mills says that the uncinate gyrus is not only the center for smell and taste, but also for the representation of the sensations produced by the activities of the abdominal and thoracic viscera. This case also had a projection of a submerged memory of a woman dressed in blue who caused her fear. These are called voluminous mental states by Hughlings-Jackson, and frequently accompany idiopathic epilepsy, but it is noteworthy that when incoördinate sensory resuscitations like this have occurred, and organic intracranial disease has been found later, that the lesion has invariably been in the temporal lobe. In a series of nine proved cases of tumor in the tempero-sphenoidal lobe, he obtained a clear history of dreamy states in seven, and a subjective sensation of special sense in eight, and the entire nine at one time or other in their history had one or the other phenomenon.

Motor Area. A tumor of this area may be cortical, and give rise to very definite symptoms, since the functions of every part of this area are so well understood, or it may be so far down in the central portion that its only effects will be an irritation of the motor tracts. The latter localization will produce a progressive localized paralysis without preceding spasms. If it be cortical there will first be symptoms of irritation followed by those of destruction, i. e., spasms followed later by paralysis. The first irritative symptom will be tingling in a definite area, but this may be replaced by a numbness. This is a signal symptom, and indicates the location of a growth much more clearly than the more pronounced symptoms of a later date. By reference to charts of the brain, the growth can be easily localized when in the motor cortex, which is not true of tumor in other localities. As the growth increases in size the intensity of the irritation becomes greater, and spasms replace the paresthesias, but however great the convulsive condition becomes, there is a precursory numbness or tingling which constitutes a sort of an aura, always of great localizing significance. The spasms at first are of slight intensity, and do not disturb consciousness, but are, from the first, followed by a temporary weakness which in recurring attacks becomes a temporary paralysis. Finally the slight initial spasms develope into tonic or clonic convulsions which recur at intervals, limited, or beginning upon one side of the body, and after several repetitions are succeeded by permanent paralysis. If a tumor is small, the spasm may occur again and again, and finally eventuate in a paralysis without a loss of consciousness at any stage. The order of the evolution of the condition is very important, since a general convulsion of sudden onset, and accompanied by loss of consciousness, is not a sign of tumor, unless the preceding symptoms have been present. If there is a permanent paresthesia, some ataxia with the paralysis, or a marked incoördination, or disturbance of the muscular sense, we are justified in localizing the growth in the motor zone behind the fissure of Rolando. Aphasia and agraphia would be produced, if the growth was in the lower portion of the motor zone, in front of the fissure. If the irritative symptoms were slight at first, increased slowly, and in paroxysms, and paralysis was late and rather slight, there would be a great probability that the tumor was in the meninges, impinging only on the cortex, or that the growth was subcortical. It might be very deep, if it was a large tumor. While focal epilepsy is usually a symptom, a tumor may yet be localized, as in the motor area, by a hemiplegia of very gradual onset, without other general signs of a tumor. All these tumors produce an alteration in the deep reflexes, and one of the early signs of paralysis is a marked increase in the reflexes. Muscular twitching is a sign of impending paralysis, and at the same time of the location of the growth. The only atrophy in the muscles involved is one of disuse, and there are no electrical changes. Vasomotor disturbances and tachycardia have been symptoms.

Parietal Region. Destruction of this area gives rise to sensory symptoms in two ways, viz., directly and indirectly, but does not cause pain. Asteriognosis is a direct result of injury to the superior parietal. and alexia and visual aphasia, if it is in the inferior lobe, if the left side is the one affected. Since gait is controlled by proper sensory information, growths in this region may produce ataxia, and since the cortical representation of the third nerve is near the angular gyrus in the descending parietal, we may find a paralysis of this nerve. The indirect results may be numerous. If the growth pushes forward there will be spasms and paralysis, while if it dips downward it will irritate or cut off many of the tracts concerned in sensation. If it presses upon the tracts going to the posterior portion of the internal capsule from the rear portions of the brain, we shall find losses of both general and special sense. There will certainly be anesthesia and ataxia; if the optic tract is cut off we shall find bilateral homonymous hemianopsia (see cut on pg. 177), while a projection to the lower front portion will produce agraphia.

Tumors of the Occipital Lobes. The occipital lobes are preëminently a visual center, and therefore both compression and destruction symptoms concern the sense of sight. Since the visual tracts are long, and a growth at many points may produce similar symptoms, such losses do not have a great localizing significance. If, however, such losses are accompanied by sharp occipital pain extending into the neck, there is ground for localizing the cause in the occipital lobe. Even a small tumor will compress the tracts sufficiently to produce hemianopsia; and word-blindness will frequently accompany it. If on the left side, there will also be mind-blindness, or psychic blindness (alexia), where with perfect sight the person fails to recognize a familiar object. These losses are as frequently the result of subcortical as cortical growths. Complete blindness may be produced by a bilateral lesion. We should assume that the growth was in the front, or had extended toward the front of the lobe, if in addition to some of the before-mentioned losses, we should also find hemianesthesia, hemiataxia, and perhaps hemiplegia.

Tumors of the Sylvian Fissure, or the Island of Reil. By directly pressing upon the Island of Reil and the capsule, which lies under it, they may produce aphasia, and if deep enough, give rise to all the symptoms of a tumor of the capsule itself. Indirectly tumors of the fissure of Sylvius may press upon the tip of the third frontal, and produce motor aphasia, or by invading the operculum (parts of the frontal and parietal which overlap the insula), paralyze the face, or so damage the inferior parietal, or superior temporal, that various forms of sensory aphasia are induced.

Tumors of the Central Parts of the Brain. These are unusual, and have no certain localizing symptoms. They simulate tumors of the frontal lobes in that the patients are immobile dements, with a great tendency to stupor, and they usually terminate in coma. If we find these symptoms with a hemiplegia, with a lesser degree of paralysis upon the other side, or a double hemiplegia with an absence of cranial nerve-involvement or anesthesia, it is an indication of a central lesion.

Tumors of the Corpus Callosum. Paregrossi makes these diagnostic points: early mental disturbance, and disorders of speech differing from aphasia; various motor and topographic disturbances relating to equilibrium, absence of general tumor symptoms; absence of cranial nerve disturbances; subacute onset of the growth, and progressive character of the symptoms. Williamson makes a clinical report of a case confirming the symptomatology in the main, but says that the speech defect is due to a fine tremor of the tongue, lips, facial muscles, hands and arms, with tremulous speech and a mental impairment altogether suggesting general paresis. Bristow describes a state of stupor or somnolence rendering the patient difficult to nourish or to interrogate. This symptom was prominent in one of my own cases. Williamson's case had no headache nor vomiting until the last two weeks of life, but had marked double choked disc. There was a well marked double Babinski, and Oppenheim's reflex without ankle clonus, or marked paresis of limbs.

Tumors of the Lenticular and Caudate Nuclei, and the Optic Thalamus. These are fairly common, and are principally marked by the general symptoms of tumor, and paralyses resulting from encroachment upon the internal capsule. The functions of these ganglia are not sufficiently understood to make accurate localization possible, but diagnosis between growths in these bodies rests upon the fact that tumors of the lenticular and caudate nuclei are more apt to produce motor palsies, while those of the thalamus will give rise to a loss of the power to recognize the position of the limbs of the opposite side. Hence there will be an awkwardness of movement, the limbs will assume strange positions, and there will be athetoid movements. We may also see hemichorea. Some of these losses depend upon the disturbance of the cutaneous and muscle-sense. Lesions of the thalamus cause burning, smarting pains. If the growth is in the pulvinar, there will be optic neuritis at an early date, while if it extends into the posterior part of the internal capsule there will be hemianopsia, and, less often, hemianesthesia. If it projects into the lateral ventricle, there will be symptoms of distension. A tumor in the posterior portion of one thalamus invariably produces homonymous hemianopsia. The growth in the pulvinar produces a hemianopsia, which is distinguished from that resulting from a similar connection in the occipital lobe by the presence of a distinctive hemiopic pupillary reaction, or inactivity. A gradual hemiplegia, with twitchings and jactitation of the limbs preceding it is a sign of encroachment on the motor tracts.

Tumors of the Base. They often give rise to symptoms referred to the lobes which they underlie, but continuous pressure often produces a perforation of the bones, and they protrude externally, or into the cavities in their neighborhood. They often invade the orbit, nose, mouth, antrum and tympanum, and their presence is indicated by discharge of brain-tissue, blood, or the structures of the tumor.

Tumor of the Pituitary Body. The pituitary tumors have recently attracted a great deal of attention on account of the elaboration of a definite symptomatology, and the advances in the possibility in a successful attack, due largely to the work of Cushing and his confreres, upon whose data this portion of the article is based. The hypopysis, or pituitary gland, lies on the sella turcica of the sphenoid bone, and just posterior to the optic chiasm. It is composed of two portions, an anterior, and a posterior. The anterior portion, or hypopysis cerebri, is an invagination of the ectoderm of the buccal cavity, and envelopes the posterior portion, or infundibular body, which is an outgrowth of the mid-brain. Both portions have a peculiar secretion. That of the anterior lobe, if exaggerated (hyperpituitism) produces giantism or acromegaly, while under secretion (hypopituitism) produces adiposity, and physical and sexual infantilism, and this portion cannot be completely extirpated without endangering life. (Brubaker.) The posterior portion secretes a fluid which probably contains a hormone, entering the circulation by way of the cerebro-spinal fluid, which is the efficient agent in the metabolism of carbo-hydrates. The posterior lobe may be extirpated with safety to life, but the metabolism is perverted. The symptoms of tumor of the pituitary may properly be brought under three groups: 1. Such as show existing or past hyperpituitism. The striking symptom is overgrowth, resulting in giantism when the process antedates the ossification of the epiphyses (typus Launois); resulting in acromegaly when it is of later occurrence (typus Marie). 2. Those in which the clinical features of hypopituitism predominate, adiposity, with persistence of both skeletal and sexual infantilism when the process originates in childhood (typus Froelich); adiposity with sexual infantilism of the reversive form when it originates in the adult, the type explained on experimental grounds. 3. The mixed or transition type, exhibiting some features of both, and termed dyspituitarism. States of increased functional activity have with few exceptions been found to be caused by tumor; and decreased secretion, while arising at times from mere blocking the exit, in many instances is the result of the destruction of the glandular tissue by new growths.

Looked at in another way symptoms are: 1. Neighborhood symptoms. 2. General pressure phenomena. 3. Secretory or glandular symptoms proper. 4. Polyglandular manifestations. Neighborhood Symptoms: There is more discomfort when the tumor is in the gland than when it is interpeduncular and intradural, as is the case in most of the benign cases causing primary hypopituitism. Headache is usually bi-temporal and severe, and persistent when it stretches the capsule, which may be the cause of some obscure headaches. They often subside when the patient lies down, possibly due to the low blood pressure from which these patients suffer. There are: photophobia with deep orbital discomfort, and tenderness of the eyes to pressure; deformity of the sella turcica; optic nerve atrophy without previous choked disc, and in fact, from pressure, it may be a late addition to optic atrophy. The increase in size of the sella does not predict injury to the chiasm, which, however, is a common condition. Bi-temporal hemianopsia is usual, and there is some exophthalmos. Deformity of the color fields is almost certain, and precedes deformity of the fields for form. The presence of diplopia shows some oculomotor involvement. Slight nystagmus has been observed frequently, even when no occulomotor faults could be discovered. Trigeminal neuralgia occurs at times. The uncinate region may be indented, and we find characteristic symptoms, viz., dream states with gustatory or olfactory aurea (psychical epilepsy). Epistaxis is common, and a cerebro-spinal rhinorrhea was observed in one case of Cushing's.

Pressure symptoms: headache may be the only symptom, since vomiting is a very inconspicuous feature in these cases. The peculiar pressure symptoms are evidences of intracranial pressure on the veins, and are shown by the fullness and tortuosity of the veins of the scalp and the palpebral venules. Glandular manifestations: a functional hyperplasia of the pars anterior produces skeletal overgrowth. Fluctuation may occur in the production of hormones stimulating bone developement, and therefore giantism may stop at any point, and if hyperpituitism again occurs, acromegaly will supervene on giantism, but the contrary is never true. The skin becomes greasy from hypertrophy of the sebaceous glands, the hair follicles enlarge, and the cutaneous structures are generally thickened. The subcutaneous connective tissue also shares in this hypertrophy. Adiposity, when primary, is due to involvement of the posterior gland; it may be local or general. Cerebral adiposity is a term intended to convey the idea that the secretion of the posterior lobe has for some reason not been produced, or that it has been barred out of the circulation. This produces the infantile and hyposexual types. Carbohydrate tolerance: polyuria and polydipsia are often seen in some period in the disease. This observation may explain the polyuria so often seen after cranial injuries. The patients feel cold, and actually have hyperpyrexia. The blood pressure is low. They are drowsy and torpid. The tissues are so insensitive that it has been possible to operate with a minimum quantity of anesthetic, and at times with none at all. Constipation is common. The larger number of patients have shown some psychic disturbance. There may be epilepsy of a general as well as of the uncinate type.

Tumors of the Corpora Quadrigemina. These growths are rarely limited to these bodies, but usually involve the crus and pineal gland, or "vice versa." Since these bodies are the primary centers of sight and hearing, and through the crus, in addition, pass both sensory and motor tracts, the symptoms are very wide in possible range. The corpora quadrigemina are the seats of the coördination of the movements of the eyes, and therefore all reflex motions of the eyes are interfered with. Growths usually are sufficiently large to compress the oculo-motor fibres, and diplopia will result. The eyes cannot follow a light, or gaze steadily at a page, and therefore such ocular losses, combined with a reeling gait and the general symptoms of tumor, will localize it in the corpora quadrigemina. If the crus is also compressed by a growth in this locality, there will be a "superior alternating hemiplegia," that is, there will be an oculo-motor paralysis of one side, accompanied by a paralysis of the extremities of the other side. This is the syndrome of Weber, and is also said to be caused by a tumor (tuberculous or syphilitic) on the inferior and inner side of the crus, and involves also the face, tongue and limbs of the opposite side. In tumors of the quadrigemina there is loss of the pupillary reflex, and nystagmus is present.

Tumor of the Crus. If a tumor occurs here, we shall typically have a third nerve palsy of one side (which indicates the side upon which is the tumor), and a hemiplegia of the other half of the body. Extension of the tumor may induce the paralyses just mentioned, and if it compresses the optic tracts there will be bilateral hemianopsia. An intention tremor is also found, and also one similar to paralysis agitans.

Tumors of the Pons. A tumor may be so located as to give rise to few symptoms, until it attains a considerable size. The usual symptoms are a combination of hemiplegia of an alternating type, and cranial nerve palsies. Some combination is true in 85% of the cases, but some are solely hemiplegic, and a few produce lesions of the cranial nerves alone. Bilateral symptoms are more common than in lesions of the higher levels. The paralyses of the limbs will be of the upper-neuron type, i. e., increased reflexes, slight atrophy, and unchanged electrical reactions, while those of the cranial-nerves will be of the lower-neuron type, i. e., lost reflexes, decided atrophies, and the electrical reaction of degeneration. Rulably a tumor of the upper part of the pons will paralyze the third and fifth nerves, producing oculo-motor palsy, dilatation of the pupils, ptosis, tingling, pain and anesthesia of the face, sometimes ulceration of the cornea, and, at times, grinding of the teeth in sleep. A lower localization produces a paralysis of the sixth nerve, which allows the eyeball to rotate inwards. Below this it will involve the seventh nerve, and facial palsy will result, while an impairment of the eighth will cause deafness. Conjugate deviation of the eyes is toward the side opposite the lesion, just contrary to the condition entailed by a cerebral lesion (the eyes look at the lesion in cerebral destructive lesions, and away from it in pontile destruction), is sometimes an accompaniment of facial palsy, and under such circumstances the face will be palsied on one side, and the eyes will deviate to the other. Tumors in this locality are liable to exert pressure upon the middle lobe of the cerebellum, and under such circumstances there will be a staggering gait, usually deviating toward the side opposite the tumor, forced movements, and general unilateral, or alternating convulsive movements. These tumors of the pons uniformly produce a loss of knee-jerk, and the control over the bladder and rectum is frequently impaired.

Tumors of the Medulla. Here also there is a combination of cranial-nerve symptoms and hemiplegia. Irritative symptoms of the ninth, tenth and eleventh nerves are most usual, and therefore there are difficulty in swallowing, irregular action of the heart and of respiration, and vomiting, and sometimes retraction of the head and neck. The twelfth nerve is least frequently affected. There may be numbness and tingling, and if there is pressure on the cerebellum, there may be cerebellar ataxia, or the limbs may be paralyzed upon one or both sides, in both motion and sensation. The termination is usually in convulsions. If the medulla is pressed upon there is apt to be glycosuria or diabetes insipidus.

Tumors of the Fourth Ventricle. These are very rare, a great proportion occurring in the infantile period, and yet one is reported in a patient sixty-three years of age. Echinococcus is relatively common, and after that is glioma, but gliosarcoma, choroidal hyperplasia, carcinoma, cavernous angioma, encephaloid, papilloma and perithelioma have all been reported in literature. The clinical symptoms are variable, sometimes being bulbopontine, and sometimes cerebellar in character. In the first category are facial paralysis, glycosuria, dysphagia, dysarthria, albuminuria, Cheyne-Stokes respiration, and paralysis of the palate and tongue. In the second category belong cerebellar ataxia, vertigo, adiadochokinesia, a tendency to fall backward, or to make rotary movements with the whole body, and priapism. A very frequent symptom is periodical dizziness; in the case of echinococcus cyst it is the most prominent symptom, and it appears not only periodically, but also on the performance of sudden rotary movements (Brun's symptom), especially in the case of loose cysts. Mental symptoms and convulsions have also been reported. (Lafora, N. Y. Med. Jour., Nov. 18, 1911.) Vomiting is as pronounced as is the optic neuritis, and is accompanied by a vertigo which is severe, but may be intermittent. The headache, vomiting, and vertigo are all caused by the stretching of the dura, which is innervated by the 4th, 5th, and 12th, and sympathetic nerves. The irritation of these nerve-fibres is reflected to the nucleus which is the source of sensory fibres to all the cranial nerves, and thence come the symptoms referred to. Nystagmus is one of the most frequent of distinctive symptoms, and it may be various in type and direction, so that it may be elicited with the eyes in almost any direction in the different cases.

. Tumor of the Cerebellum. About one-fifth of all tumors (intra-cranial) are in the cerebellum; it is especially the site of tumors in children, and glioma is the most common form (Fraenkel). The vermis is the lobe of active symptoms, while tumors have been known to attain to a very large size in the lateral lobes without giving rise to any focal symptoms. The general symptoms of tumor are, however, developed earlier than in cerebral growths, on account of their position between the base of the skull and the tentorium; the headache is peculiarly severe, and constant, and is characteristic when it is occipital by an extension down the spine, with stiffness of the spinal column accompanying it, although its location may be frontal. Optic neuritis is early, frequent and pronounced, leading to early optic atrophy and blindness. On account of the peculiarity of position many symptoms will develope which are the result of the irritation, or pressure upon neighboring structures. It matters little whether the tumor is in the vermis, or in the lateral lobe, sooner or later the cranial nerves will be irritated and there will be tics (muscular spasms of a somewhat constant type), followed when irritation is succeeded by pressure, by paralysis of the face, deafness, disturbance of swallowing, and symptoms of vagus disturbance, and further growth will increase the deep reflexes, while the superficial or skin reflexes are unchanged. If the cranial nerve symptoms appear early, it is evidence that the tumor is on the under aspect of the vermis. If a tumor is in the lateral lobe, its increasing bulk presses upon the vermis, and its characteristic symptoms become evident, and their appearance, some time after the general symptoms of tumor, point to an origin outside the vermis. Under such conditions, the lateral irritation of the vermis may force the patient to carry his head with the ear resting on the shoulder homolateral to the tumor, the face is directed upward toward the other side, and the spine is laterally bent, with the concavity to the side on which is the tumor (Bolton). The characteristic symptoms of a tumor in the cerebellum, i. e., vermis, are as above stated, but they are bilateral. The patient will reel and sway, but will have no real ataxia unless the vermis is initially, or secondarily affected. Although the patient will sway when he attempts to stand with the feet close together, it differs from the Romberg's sign of tabes, in that the swaying is as pronounced with the eyes open as it is with them closed. If the patient is able to walk he may show a "cerebellar gait," which is reeling, swaying like intoxication, and is the result of a combination of incoördination, vertigo, and weakness of the spinal muscles (titubation). There has been a very pains-taking investigation of the localizing significance of the constant tendency to fall in one or another fixed direction for a given case, but with present knowledge its diagnostic value is slight. Irritation of the cerebellum produces movements of the eyes, usually conjoined, as single muscles cannot be innervated from that point. Nystagmus is often observed as an early symptom, and while in many cases due to irritation of the oculo-motor centers about the fissure of Sylvius by fluid in the ventricles, in other cases it is undoubtedly a direct cerebellar symptom. Attempts to look upward cause vertigo. This is a valuable sign of cerebellar disease when taken in conjunction with other symptoms. The vomiting and headache observed in all cases is due to irritation of the vasomotor and pneumogastric centers in the floor of the 4th ventricle just under the cerebellum.

Paralysis is always a secondary symptom, and is shown not only in the trunk and limbs, but is very common in the external recti, causing bilateral or unilateral deviation of the eyes. As the tone of the muscles on one side of the body is furnished from the cerebellar lobe of that side, it is natural that defects referred to tonus should be a prominent symptom of cerebellar disease. It is described under various names. Babinski points out what he terms Adiadochokinesis, and says that it is the most valuable diagnostic sign. By this term he indicates a slowness in movements of an alternating character, and this is best shown in the successive pronation and supination of the forearm and hand. In lesions of the cerebellum there is a slowness on the homolateral side, and it may be shown in the speech as well, since the patient cannot readjust muscular tension in the lips and tongue readily enough, and therefore staccato speech is one of the forms of defect resulting. Babinski finds also that there is a fault which he terms hemiasynergia, which consists in a loss of the power of smoothly executing what might be called compound motions. If a normal person be asked to flex the leg upon the thigh, and the thigh upon the abdomen, and then is told to straighten out the member, it will be done in one compound motion, while the victim of hemiasynergia can only accomplish this in two motions. Hemiataxia, hemiasynergia, and tremor point out the affected side, as will also an atonic paralysis. Jackson believes that a destructive lesion of the vermis causes paresis or paralysis which is most marked in the muscles of the trunk, next in the lower extremities, and least in the upper extremities.

Tumors of the Cerebello-pontile Angle. This site is the area between the lateral lobe of the cerebellum, pons, and medulla. The tumors of this area are of peculiar interest, since they are fibromata on the nerves, and if discovered early their removal results in actual cure, but this happy result cannot be expected later. They arise especially upon the 8th nerve, but may invade initially or in order the 7th, 6th, and 5th nerves. Typically the 8th being affected, the initial symptom will be diminution of hearing on the same side, with buzzing, rushing, ringing noises; later total deafness. Often this will be disregarded, and cerebellar incoördination will be the first complaint, or there will be compression symptoms of the structures in this locality. These tumors show a predominance of unilateral focal signs on the part of the 5th, 6th, 7th, and 8th nerves, while tumors of the pons and medulla show lateral signs with alternate hemiplegia.

DIAGNOSIS. In spite of advances in data for localization, it is clear that the evidence upon which a diagnosis must be made is still in many cases so confused and contradictory that a satisfactory localization is impossible. The X-Ray, in spite of great advances in technique, is still of dubious value, as a rule, in the localization of, or in the determination of the nature of an intracranial growth. It is valuable, however, in the highest degree in the corroboration of the diagnosis of tumor of the pituitary body. The enlargement or deformity of the sella turcica, or rarefaction of the portion of the sphenoid bone beneath it is due to the existence of an enlargement, almost certainly tumor of the gland. There are three phases in which the diagnosis of tumor must be considered. Is there a tumor? Where is the tumor? What is its nature? We should diagnose a tumor from the presence of the following symptoms, viz.: a constant and quite severe headache, vomiting of a cerebral character, double optic neuritis, retardation of the pulse, generalized spasms, somnolence, mental dullness, and vertigo. But more briefly, we may reason that while headache is common to many diseases and conditions, if papillitis, or double-choked disc is also present, the cause of the headache is almost certainly tumor. Even if a person has headache with papillitis, the presence of an accompanying chlorosis should cause us to be guarded in making a diagnosis. If, however, we can add to headache and choked disc, projectile vomiting and vertigo, the diagnosis is certain.

DIAGNOSIS OF SITUATION. For this element in the diagnosis it is necessary to carefully separate the general from the focal symptoms, and to assure ourselves of the order of their appearance. When the general symptoms are pronounced, and the focal symptoms are indefinite, or altogether wanting, there is a strong probability that the growth is in the frontal or temporal region of the right side, or is in one of the lateral lobes of the cerebellum. The combination of the symptoms may be as characteristic of their location as is their order, or mode of extension. This is especially true of tumors of the crus, and the corpora quadrigemina. When a tumor is in such a position that it exerts pressure upon the large vessels, or interferes with the general circulation, it gives rise to symptoms indicative of thrombosis. The symptoms may be direct, or indirect.

NATURE OF THE GROWTH. We now come to the third factor in making a diagnosis. When we are so fortunate as to be able to localize it, we have, from that fact, some grounds for a surmise as to its nature.

Tubercle and glioma are more liable to be found in the pons and cerebellum (Glioma rare, Fraenkel), while sarcoma is frequent at the base, and outside of it on the walls. Syphiloma is common in the pons, basilar area, and the cortex, but is rare in the white-matter of the brain and the cerebellum. Fibroma and glioma are interstitial growths, and favor the deep structures, while tumors of the ventricles and meninges are liable to be sarcomata. The stage of life also bears upon this subject, since in childhood we are apt to find tubercle, glioma and sarcoma, while adults are more liable to develope the harder growths and syphiloma, while carcinoma is commonly confined to an advanced period of life. The clinical history is another element in the formation of our opinion. A rapid onset followed by a period of stasis, especially if the patient be in the first half of life, indicates that the growth is tubercle. Glioma and sarcoma are steady and insidious in their rate of progress, while syphilis is rapid in onset and course. Apoplectic seizures are an indication that the growth is a glioma, since such tumors are most frequently subject to hemorrhage. The condition of other parts of the body is a strong indication of the variety of morbid process going on in the brain. Syphilis, cancer of the breast, or of other regions, actinomycosis of the jaw, and hydatids of the liver strongly indicate that a growth of similar nature is in the brain.

DIFFERENTIAL DIAGNOSIS. Chronic Diffuse Nephritis, or Contracted Kidney have points of similarity, but nephritis in any form may be distinguished by the history, by examination of the urine, and the state of the heart and arteries.

Profound Anemia in young girls may give rise to all the general symptoms of tumor, but it lacks the focal symptoms, and examination of the blood gives a different picture; under any circumstances, relief of the anemia cures the case.

Amenorrhea may produce all the symptoms described in speaking of anemia, and no anemia may be present, but if the menstrual function be restored, the diagnosis is at once cleared up. If an ocular defect is added, the diagnosis from an early stage of tumor may be difficult, for Diller reports a case which gave the clinical signs of tumor. viz., vertigo, headache, projectile vomiting and diplopia, but without choked disc, which was relieved entirely by correction with glasses. It was noticed that a preliminary instillation of atropine gave relief, which is a hint for diagnosis. A slight degree of choked disc may be present.

Chronic Lead Poisoning. Bramwell says that he never makes a positive diagnosis of intracranial tumor until he has positively excluded lead poisoning, differentiated upon the following grounds: lead is indicated by a blue line upon the gums, anemia, intestinal colic, constipation, and wrist-drop preceded by tremor. There is a previous history of the ordinary symptoms of lead poisoning, the occupation of the patient favors its existence, and there is evidence of lead in the urine.

Tabes. This may resemble cerebellar tumor superficially, but the knee-jerks are never increased in tabes, the vomiting, if present, is with the pain of gastric crises, and the static ataxia is aggravated by closing the eyes, while it is not so influenced in cerebellar tumor.

Hydrocephalus may be the result of intra-uterine disease, meningitis later in life, or arise as a result of the presence of tumor in the cranium. It will give rise to pressure symptoms, and in some cases closely resemble pontile tumor from the fact that as the pressure increases the crus and pons are displaced, and the cranial nerves suffer. Such symptoms with a paralysis of the lower limbs would lead one to think of a tumor of the base. The diagnostic point is that there is a spastic paralysis, but that it is bilateral, and developed without previous spasms; there is also the difference that the lower limbs are affected more than the upper, and there is incoördination of the hands. The child has scissors-leg. Lumbar puncture shows more albumin in the cerebro-spinal fluid of tumor than in hydrocephalus.

Chronic Cerebritis or Encephalitis may simulate a tumor very closely, but the cases are very rare.

Acute Encephalitis gives rise to an optic neuritis, but it differs in being an acute febrile disorder, and differs also in its later history, in the fact that it is usually fatal in a few weeks, or terminates in recovery in a somewhat longer time.

Meningitis, acute or chronic, is comparatively rapid in its developement, as compared with the months which must elapse in the developement of the most quickly growing tumor. Meningitis causes a neuro-retinitis, while a tumor produces choked disc. In meningitis the paralyses are bilateral, while they are unilateral in tumor, unless it is multiple. The paralyses of meningitis are slighter and more variable in their duration, and there is fever, marked variation in the size of the pupils, a slow and hard pulse, constipation, retraction of the abdomen, and vasomotor symptoms. Certain diagnosis can only be made in the tuberculous forms when the case of meningitis is one of those which developes rapidly, is complicated by hydrocephalus, and is fatal in three or four weeks. In general it may be said that the headache of meningitis is more continuous and severe, that a neuro-retinitis is less rapid and intense than the choked disc of tumor, and that a general subsidence of symptoms with recovery would indicate a meningitis rather than a tumor. If the meningitis is at the base, certain diagnosis is impossible, since a tumor is often complicated by a meningitis. A chronic meningitis of the base especially imitates a tumor of the sella turcica, since the same cranial nerves will be implicated in both conditions, but the meningitis produces a descending optic neuritis without coincident symptoms of an increase of intracranial pressure. When in young children a diagnosis must be made between tuberculous meningitis and tumor, we should lean toward the former if with intense hyperesthesia to light and sound there was blindness with a premature closure of the fontanelles, while an enlargement of the head would point to tumor. In general it may be said that diagnosis is very difficult unless we exclude tuberculosis by the tuberculin or Chalmette test, and that this, therefore, should be the rule. The other forms of meningitis must be differentiated by the more or less hypothetical data given above, and the presence in meningitis of leucocytes in the cerebrospinal fluid. The deductions from leucocytosis must be corroborated by other symptoms, since there is often meningitis accompanying tumor. Hildebrand says that a serous meningitis is at times indistinguishable from tumor, and that the so-called pseudo-tumor is a serous meningitis with blindness and internal hydrocephalus.

Abscess. A latent cerebral abscess presents all the symptoms of tumor, which in fact it is, but with a better prognosis. Diagnosis must often be made from its etiology. Abscess occurs after severe injuries, or suppurative conditions of the ear, nasal cavities, orbit, antrum or cranial bones. After a severe injury of the skull, we may find either a tumor or an abscess, and it is never proper to assume it is a tumor if a suppurative focus is present. Unless the abscess is encapsulated, and still uncomplicated by cerebritis, meningitis, or sinus phlebitis, the symptoms appear in more rapid succession, and with greater severity than in tumor, and with fever. After a severe range of symptoms there may be a period of latency, after which there is a new exacerbation, which leads to a fatal issue. Tumors are marked by a constant accession of new symptoms, which is not true of abscess. While the headache is more severe and paroxysmal in tumor, a unilateral localization is rather a sign of abscess. Optic neuritis is more common with tumor, and mental changes are more gradual and constant.

and the local symptoms appear more slowly. The temperature is normal, or subnormal, in both, but if we find sudden fluctuations of temperature with sweat, abscess is the more probable cause. Marked emaciation is more often a characteristic of abscess, but it occurs in an unaccountable manner in some tumors. Intra-cerebral hemorrhages are a common incident in tumor, while meningitis is the usual complication of abscess. Symptoms of sinus phlebitis indicate abscess. A duration of one or two years with no accession of new symptoms, indicates abscess. After such a period there can be no doubt, unless the tumor is situated in some latent area, and so has attained a considerable size without marked symptoms. Hemorrhage would then differentiate, unless it could be explained by an injury, or some degeneration of the arteries.

Cerebral Hemorrhage or Softening. In such cases an examination of the patient will show evidences of constitutional disease, and his history will reveal confirmatory data. The symptoms after the hemorrhage will be diagnostic if an adequate history be wanting. If a hemorrhage has resulted from arterial degeneration, the symptoms will decrease after a time, and there will be no optic neuritis, but if the hemorrhage has arisen from the rupture of vessels in a tumor, the symptoms will show a steady increase after the hemorrhage, there will be headache, convulsions, and optic neuritis.

General Paresis. Ordinary cases show no similarity, but some doubt may arise when it begins with persistent headache, and paralytic and irritative symptoms are present, whose exact nature cannot be identified without accessory symptoms. Tumor of the medulla may present a quite similar symptomatology. Choked disc does not occur in paresis, but it is replaced by pupillary rigidity. The mental state of tumor is stuporous, while that of paresis is some grade of grandiose delusions, or a definite melancholy, going on to dementia. Paresis has apoplectiform attacks, which resemble cortical epilepsy from tumor, but the result is a decrease in intelligence, rather than the paralysis occurring in tumor. Tumor at the base may be marked by tremor and disorders of speech, but such growths do not produce the combination of a tremor of the lips, upon strong retraction, with slurring speech.

Epilepsy has convulsions similar to those occurring in the history of tumor, but with differences. The convulsions from tumor have a clonic stage followed by the tonic, while the opposite order obtains in epilepsy. The convulsion of epilepsy lasts for two or three minutes only, while that of cortical or Jacksonian epilepsy (the variety found in tumor) lasts many minutes, and it may be hours. An epileptic convulsion is terminated by a deep sleep of a half-hour, to one or two hours' duration, while that is replaced in focal epilepsy by a slight state of coma. The convulsion of the epileptic may leave him mentally dull for some hours, but he does not suffer from the severe headache, the projectile vomiting, or vertigo, neither does he show choked disc.

Tumors of the uncinate gyrus, or those impinging upon it, have been noted in a previous section as competent to produce epileptiform convulsions of both a psychic and a motor type.

Migraine. This may have a severe headache followed by a choking of the disc, in rare cases, and the pulse may be retarded, but the condition is a periodic one. Some cases of migraine become constant after a time, but the disc will not show choking under such conditions, and further and distinctively, the disease presents the same group of symptoms without much variation, while a case of tumor is notable from the constant accretion of new symptoms.

Disseminated Sclerosis. Tumor may present such symptoms that the diagnosis is doubtful if it be located at the base, since a tumor in the cerebellum, pons, corpora quadrigemina, or uncinate gyrus, may give rise to nystagmus, tremor, headache, vertigo, and an uncertain gait. It will be especially confusing if the case is one in which choked disc has reached its final stage, and produced optic atrophy, which is a symptom of disseminated sclerosis, but with a difference, as the typical optic atrophy of multiple sclerosis affects only the nasal halves, giving rise to the distinctive bitemporal hemianopsia. The points of difference are these: the tremor in tumor is finer and less volitional; just as often passive as intention; indefinite; it is also accompanied by ataxia, while the tremor of multiple sclerosis is accompanied by spasticity. We rarely find a case of disseminated sclerosis which does not develope spinal cord symptoms while in it we do not find stupor, retardation of the pulse, vomiting, aphasia, cortical epilepsy and spasms.

Hysteria may so resemble the symptoms of tumor, that the diagnosis must depend upon exclusion. The headache of hysteria may be as violent as that of tumor, but it is characteristically aggravated if attention is directed to it. This is also true of neurasthenia. If optic neuritis is developed, its presence will clear up the diagnosis, since this has never been seen in hysteria. It should be remembered that the presence of a tumor may induce hysteria in a favorable subject. In two cases large tuberculous tumors gave rise to symptoms which were taken for major hysteria.

Intra-cranial Aneuyrism. This is, to all intents and purposes a tumor. It may sometimes be diagnosed from the presence of a bruit, or the bone may become so eroded by its pressure that pulsation may be made out, or it may invade one of the sinuses, giving the symptoms of sinus thrombosis. It should be thought to be a possibility if there has been syphilis, general arterial degeneration, or an injury to the skull.

Alcoholism. Chronic alcoholism may produce many of the symptoms suggesting the presence of a tumor. There may be headache, apoplectiform seizures, vomiting, and even optic neuritis. This latter condition furnishes a ground for differentiation, since the optic neuritis from alcohol has a central scotoma for colors. Usually there are the characteristics of gastritis, diarrhea, and a general tremor of the body, limbs, lips and tongue without any in the face.

Multiple Neuritis. This, whether from alcohol or a cachexia, may produce an optic neuritis, but if the limbs are weakened by a tumor, the deep reflexes are exaggerated in place of the diminution or loss found in multiple neuritis.

Prognosis. This depends upon the age of the patient, the kind of growth present, and its location. The prognosis for duration of life, and return to health, is better in the child than in the adult. The compression symptoms are not so severe, since the cranium is more elastic, and the sutures separate more easily, and children are more liable to be affected by tubercle, which may be operable, and is thought to be more liable to remissions, and sometimes spontaneously absorbs. The average length of life of a patient giving signs of the presence of a tumor is from two to four years, but some cases die in a few months, and some have lived as many as ten, or even fourteen years. A sudden fatality is always imminent, but remissions often occur, and have been known to endure as long as eight years, and many cases have a period of latency, after which there is renewed growth. Another method of spontaneous relief of a temporary character is by the perforation of the bones by the growth. When this occurs, there is a discharge of the cerebro-spinal fluid, often through the nose, which temporarily relieves the intra-cranial pressure.

Different varieties of growth give rise to some degree of difference in the prognosis. Solitary tubercle, glioma and the hard varieties of sarcoma are of slow growth, cholesteomata and cavernous angiomata are especially chronic. The soft sarcomata, and carcinomata are rapidly fatal. Taken as a whole the prognosis is best in syphiloma, but they are prone to return. Echinococcus cysts, and cavernous agiomata, and solitary tubercle, are of relatively good prognosis. Diffuse tubercle (as opposed to the solitary form) and syphilis may have a bad prognosis, since they are apt to be complicated by meningitis. Any form of tubercle and carcinomata has a bad prognosis, since it is usually secondary to like conditions in other parts of the body. Any growth which is hemorrhagic has a grave prognosis, since there is a great liability to apoplexy. This hemorrhage may be into the brain about the growth, in which case it has the usual prognosis, but it may be into the growth, and affect the brain only by increased compression. In this latter case the initial fall of temperature peculiar to cerebral hemorrhage is said to be absent. Spasms indicate cortical irritation, and paralysis destruction of the motor cortex, or of the motor paths.

Favorable symptoms are a subsidence of the headache, and a recession of the optic neuritis, and if the optic neuritis developes slowly, it indicates a like rate of developement in the growth. Symptoms of bad omen are a stuporous mental condition, convulsions, severe vomiting (both from its causation and also its effect upon nutrition), a rapidly developing and severe grade of optic neuritis, apoplectiform seizures, and, above all others, bulbar symptoms. Aside from syphilitic tumors, which are sometimes amenable to remedies, the prognosis depends upon the possibility of removal of the growth by surgical means, and a tabulation of recent statistics indicates that there is a possibility in from five to nine per cent. Practically the only tumors capable of removal are those which are cortical, or closely subcortical, in the motor zone. This arises from the fact that they are accessible, and give rise at an early date to localizing symptoms, and can be removed before they attain a great size. Large tumors have been successfully removed, but the probability of success is less. If small they may be removed, even if of the diffuse variety, but when large they are operably only if they have a capsule, or are in some way circumscribed. Multiple tumors are always inoperable. In some cases this rule is inapplicable, since some diffuse tuberculous and syphilitic growths have been excised with a favorable result. Aside from growths of the motor zone we should be justified in operating over the third frontal on the left side, if in addition to the general symptoms of tumor, there had been from the beginning some degree of aphasia. Under the same general conditions we might operate over the occipital lobe, if there had been an early and constant hemianopsia, and over the temporal if the loss had been one of audition. Cerebellar tumors have been successfully operated, but it is very exceptional. Forty-eight cerebellar tumors were operated upon with 20 deaths, 16 recurred, and 1 was apparently cured. Cerebellar cysts have a good prognosis. Tubercle is operable at times, even when complicated by meningitis. Basilar pontile, and medullary regions are outside the possible field. Carcinoma is the least operable of all. Psammomata, cholesteomata, angioma, dermoid cyst, and the parasitic form are operable if they can be localized. Sarcomata can usually be enucleated, tubercles and fibroid tumors shelled out, but gliomata are infiltrating, and cannot be completely removed, and are liable to recur from the remnants. Cysts and old abscesses are readily drained. If the growth is in the lateral lobes, localizing symptoms are absent, and if in the middle lobe it is inaccessible.

Death results from cachexia, induced by pressure and consequent inhibition of cerebral functions, or from hemorrhage, convulsions, exhaustion, infection, bulbar symptoms, or intercurrent maladies.

TREATMENT. In the present stage of diagnostic knowledge and operative technique about seven per cent. of tumors are removable. The hope of a beneficial result, both for the preservation of life, and also of function, rests upon early removal. Horseley says that operation must be done upon detection of slight sensory losses, as it is too late when motor losses can be discovered. He also says that decompression may cure a glioma. Eiselberg states that trephining relieves many tumors, and that a cerebellar tumor was thus relieved for eight years. After some operations a paralysis affecting one side or a limb may result, but it always subsides later, and in most cases permanently disappears. Early removal cures tumors of the pontile cerebellar angle, as they are non-malignant in type, and also the cysts of the cerebellum. In conclusion it may be said that removal should be as early as possible if the tumor is operable. If it can be identified as a gumma, antisyphilitic treatment has promptly cured cases which appeared to be in a desperate condition. If the growth is tuberculous it is possible that iodoform may produce as favorable a result as it sometimes does in growths upon the peritoneum and meninges. The method employed by Dr. Sidney F. Wilcox of New York in those cases is worthy of a trial upon tumors. Shave the head, and once a day rub in a liberal quantity of the following preparation: Iodoform, 2 drams, Ether, 2½ ounces, Sweet Oil to make 8 ounces.

If these measures are not indicated, bi-temporal decompression should be done for the relief of the general symptoms.

THERAPEUTICS. In inoperable cases we have slight grounds for hope from remedies, but the following have been advised: The results from the administration of Thuja Tr. have been so striking that it is possible that we may find benefit from its use. Arnica, Arsenicum, Arsenicum iod., Baryta carb., Belladonna, Calcarea carb., Carbolic acid, Conium, Crocus, Gallium in substantial doses, Graphites, Hydrastis, Kali hyd., Lachesis, Nitric acid, Phosphorus, Sepia, Silicea.

When the pain in the head is excessive, it will be found necessary to invoke some one of the analgesics, and morphine may be found to be the only one of much utility.

CHAPTER XIII.

SYPHILIS OF THE NERVOUS SYSTEM.

Syphilis may affect the nervous system, at any period of its activity. Five to ten per cent. of syphilitics develope some form of nervous syphilis at a period varying from six months to thirty years from the primary infection.

SEX. Males develope it more frequently than females, probably on account of the greater vicissitudes and irregularities of the masculine life.

ETIOLOGY. The exciting cause of a localization of the virus in the nervous system seems to be anything which lowers the nervous resistance of the individual. However true it may be, it has been said in this connection that among the later results the physical laborer developes a tabes, while the brain-worker will become a paretic (denied by Nonne). It is certain that excesses, sexual and alcoholic, and traumatism have seemed in some cases to have lain in a causative relation to the invasion of the nervous system. The effects of vigorous antisyphilitic treatment of the initial and secondary stages of the disease are somewhat questionable in their influence in accelerating, or in being the essential elements in producing the subsequent tabes and paresis. Collins and Dana have given some support to this view, but Nonne denies its truth. Personally, I have found a striking absence of a history of secondary symptoms in cases which afterwards developed tabes, and the records of the London Hospital tell a similar story. Many of my cases have given a convincing history of a long and vigorous treatment of the primary infection. These facts raise these questions: did these persons have a light infection, or does vigorous treatment smother secondary symptoms; and are secondary symptoms a sort of safety-valve, the smothering of which tends to an outbreak in the future of a much more serious train of effects? The deduction is not as yet a certain one.

PATHOLOGY. The pathological changes of syphilis, according to Nonne, are grouped in three great divisions: syphilitic overgrowth, chronic hyperplastic inflammation, and disease of the blood-vessels. Necrosis of nervous tissue is only a secondary process. Besides these three forms there is a degenerative process of a varied type, to which syphilis stands in a causative relation, but it is not actually specific. In typical form it is a primary degeneration referred to under the name of parasyphilitic disease. The earlier manifestations of syphilis of the nervous system are exudative, and are most severe in their effects; during the middle period the pathological changes are very varied; and are the result of vascular changes; while the later attacks are from a primary lesion of the nervous tissue, neuron, and appear from about the twelfth to the fifteenth year of the infection. At a time not very remote from this the situation was much confused, on account of the impossibility of proving that an identical virus was responsible for the primary, secondary, and tertiary manifestations, and those later diseases of the nervous system which seemed also to be the result of the activity of the same virus. The failure in identification resulted from the fact that no microörganism could be isolated, and proved to be the cause of syphilis; and also because remedies which were helpful in these earlier manifestations were impotent for the cure of the later manifestations. The theory of a parasyphilitic virus was elaborated to explain these manifestations, but such a proposition carried with it no indication for treatment. Finally Schaudin and Hoffmann discovered the spirocheta pallida to be the cause of syphilis, and later Noguchi and Moore found the spirochetæ in the brain of the paretic, and Noguchi found them in the cord of the tabetic, and the fiction of parasyphilis was at once swept away, and the possibility of a cure for the so-called parasyphilitic diseases became evident.

Inflammation may have been so slight that the evidences of its existence are absent, but it is an incident of all syphilitic processes in the nervous system. The blood-vessels are very susceptible to the virus, and inflammation of the intima results in infiltration of it by the round cells, which are also extruded, and their organization with connective-tissue cells in combination is the origin of gumma. Nonne states in substance, that the following pathological changes occur: gumma appear as smaller or larger, single or multiple growths, or as a diffuse and extensive infiltration. They vary from a hempseed to a walnut in size; at times extraordinarily larger. The favorite location is in the dura, on the convexity, as well as at the base. At the base they appear as small tumors, with a preference for the neighborhood of the arterial trunks. When they appear in the substance of the brain, they are in the cortex, and in the region of the central convolutions. There they may so infiltrate the brain that its bulk is so slightly increased as to give very little evidence of their presence. They are sometimes in direct contiguity with the meninges, and when in the interior of the brain are not infrequent in proximity to the large ganglia. They also occur in the cerebellum, pons, and medulla, and in the hypophysis.

Meningitis occurs in all three of the meningeal coverings. The dura may have become implicated in the previous disease of the bones. Where the meninges are primarily affected, we have gumma as an infiltration, or sprinkled over the surface in a miliary form, and they produce the same effects as do gumma elsewhere. Meningitis of the gummatous type is almost always hyperplastic and fibrinous. In the great majority of cases the pia is the starting point, even when all three membranes are affected. Cases where the pia alone is affected are by no means rare, and the arachnoid may be affected alone. The leptomeninges are almost without exception thickened and adherent, and with or without a gummatous deposit. Meningitis simplex, without gummatous infiltration, may be possible, although there is no uniformity of opinion. Meningitis is usually rather generally distributed over the base or convexity, but it may be localized, which is rare. The base is the usual site, and few cases of syphilitic meningitis of any locality are found without some inflammation of all the membranes at the base. Meningitis of the base localizes especially in the interpeduncular space, and about the chiasm. The exudate in the specific basic type rulably involves the whole base, but may be only an isolated patch. The vessels suffer from compression, or an extension of the activity of the virus to their walls. The nerve-trunks may be directly compressed, or compressed by invasion of their sheaths, or by formation of a scar, or by proliferation between the nerve-fibres, or the nerves may be injured by an infection of their arteries.

Diseases of the Blood-vessels. The majority of nervous symptoms in nervous syphilis are caused by arterial degeneration. The point of initial arterial infection is in the vaso-vasorum, and the perivascular lymph spaces. Endarteritis does not occur in arteries which do not possess vaso-vasorum, and endarteritis is so often found in the brain for the reason that even very small arteries here are so supplied, while arteries in the extremities do not have them. The vessels, including the veins, may become affected by syphilis in a purely mechanical manner, both in the meninges, and in the nervous tissue itself. Secondly: they may become involved in a syphilitic process in the vicinity, i. e., the gummatous or inflammatory process attacks the vessel walls by extension, and may present a peri-, meso-, and panarteritis, with or without gummatous symptoms. Thirdly: an inflammation of the capillaries, produced by the toxin of syphilis leads to an inflammatory affection of the walls, involving primarily the media and adventitia; secondarily the intima becomes involved, and it may then proliferate further, independently. This is the picture of the endarteritis of Heubner, with the narrowing and obliteration of the lumen, and is the form most commonly found. This variety of endarteritis may be accompanied by gummatous infiltration. Fourthly: syphilis tends to produce an atheromatous degeneration of the arteries, one form of which degeneration cannot be differentiated from the ordinary atheromatous changes; the other, the arteritis of Doehle and Heller, should be regarded as extremely suspicious of syphilis. Aneurysm is not as common in syphilitic arteriosclerosis as in the ordinary type.

SYMPTOMATOLOGY. While many of the lesions of syphilis are diffuse, arteritis, gumma, and meningitis are more or less present and operative at all times, and the cases are most profitably considered under the divisions of syphilis of the brain, the cerebro-spinal type of syphilis, and the spinal, syphilis of the nerves, and the late degenerations, tabes and paresis, which have been termed parasyphilitic.

Oppenheim states that multiplicity of symptoms, their incompleteness, and their abrupt appearance, disappearance, and recurrence, are peculiarly characteristic of syphilis of the nervous system. It results from the fact that there is no other disease in which there is a pathological process like gumma, where there is a rapid growth of a neoplasm, regression of the scarcely formed structure, and a new one almost immediately formed upon its site. The brain is the most common point of attack, as the meninges of the brain are peculiarly susceptible to syphilis, and the disease may affect the brain substance also by contiguity, from gummatous infiltration, compression, and from the fact that the vessels in the pia are not only distributed to the cortex, but also to the medullary tissue underneath. The arteries of the brain, as has been said, are also peculiarly subject to endarteritis. In the early stages of the disease, within two or three years of the original infection, and in young persons, we find cases which present a symptom-complex which is the result of the invasion of the circulatory system of the brain, and is not complicated by a meningitis. Headache is the notable initial symptom, perhaps appearing long before any other, and it is characterized by being diffuse, of moderate severity, intermittent, deep-seated, increased by mental effort, growing worse at night, or being serious only in the night. Insomnia is pronounced. The headache is generally accompanied by vertigo and vomiting, and the patient is anemic and neurasthenic, and mentally changed, and irritable. He may show a jealous rage, and become forgetful, but not to such a degree as in cases of tumor. When thrombosis occurs, we find symptoms of cortical irritation in the form of muscular spasm, Jacksonian epilepsy, or numbness in a limb, or part of a limb, or a transient aphasia. If the lesion is in the occipital lobe, there will be flashes of light in the opposite half of the visual fields; while if it is in the temporal lobes we are apt to have the sound of bells, or crashes of sound. Epileptiform convulsions may be an early symptom, and the third nerve nuclei are often attacked. If softening ensues, or there is rupture of the thrombosed artery, paralysis replaces the irritative symptoms to which we have just referred. If the basilar artery is thrombosed, a fatal result is almost certain. Sometimes this process may affect a cerebral artery of considerable size, and rupture may ensue, with the production of an apoplexy of ordinary symptomatology.

The most common form of syphilis of the nervous system is a gummatous meningitis of the brain, which is more apt to be located at the base. in the interpeduncular space than on the convexity. Meningitis of the convexity may not develope any localizing symptoms, because the arteries here are small, but if present they are those of irritation and destruction of the motor cortex. Headache is the most certain symptom, and may precede all others for a long time. It may be localized or diffuse, dull or severe, and it is apt to be worse at night, but not so universally so as has been indicated in the text-books. Sometimes there is a local tenderness to percussion, which is an indication of its superficial localization. There may be no vomiting or convulsions, although spasms of a single part and monoplegia are not uncommon. The headache may be so severe that the patient is apathetic and stupid, and unconscious at intervals. There is apt to be vertigo and swaying in station, and a clumsiness and awkwardness in movement. The swaying in movement may be due to lack of attention owing to mental dullness. The preponderance of gumma may give rise to the classical picture of brain tumor. The headache may be the only symptom, and if persistent should arouse suspicions of syphilis. All sorts of pupillary anomalies may be found, and they are not always due to disease of the cranial nerves or their nuclei, but frequently are the result of degeneration of the cortex. Choked disc may appear, but is more common in meningitis of the base. Jacksonian convulsions are a symptom, and these are convulsions with retained. or only slightly disturbed consciousness. They are of one part, or a small area of a part, as a finger, or hand, but by repetition they are apt to extend until they involve the whole body. They can be localized only when paralyzed or paretic parts become involved in the spasms. Convulsions of the type of true epilepsy may be induced by a tumor formation in any part of the brain. Epilepsy is a common incident, and hemi-epilepsy is seen in a large proportion of cases. Paresthesias of all kinds may be observed. Cortical speech disturbance, clumsiness in speech, and paraphasias are common and often transient, but true aphasia of a persistent type is found only with hemiplegias, or evidences of paralysis of the face and extremities. Hemianopsia may occur from injury to the tracts, and also to the cortex. The mental changes are very characteristic, being initially stuporous and apathetic. There is a lack of interest in everything, and an inattention to bodily processes and movements, all tending to a progressive dementia. In some cases there is an initial irritability, going on to delirious states, or the delirium is ushered in by convulsions.

Meningitis of the Base. Owing to its proximity to so many important structures, a meningitis in this locality, even if small in area, is very destructive to the functional efficiency of the patient. Not only that, but the fact that the blood-vessels and lymph-channels go into, and come out of the brain at this point makes conditions for the developement of microbes peculiarly favorable. The basal ganglia, optic chiasm, pons Varolii, and the cranial nerves can hardly be expected to escape damage. If the arteries are infected or compressed, thrombosis will be the result, and softening will occur. This is most frequent in the basilar artery, and spots of softening are produced in the pons and medulla, usually in combination, and usually also bilaterally. The cranial nerves are compressed or infected, leading to their degeneration. Syphilitic meningitis of the base is ushered in by intense headache, which may be the only symptom for a long time. Next come third nerve symptoms. Strabismus leading to diplopia is most common, but ptosis is also frequent. The olfactory nerve has been found to be degenerated, and this would be more frequently discovered if lesions of it were more often sought for. There are almost invariably changes in the optic nerve. The optic chiasm is often attacked, and also the nerve. This is primary by gummatous invasion, or arteritis, or more often compressed by a gumma. We may find diminution of sight, hemianopsia (homonymous is more frequent than heteronymous), or complete blindness, which results from optic neuritis, choking of the disc, with a termination in optic atrophy. The fourth is rarely affected, and then only with other nerves, while the sixth nerve is quite frequently involved. The fifth nerve is rarely affected, but when so is more apt to be degenerated on its sensory than its motor part, and is usually affected in combination with the optic, oculo-motor, abducens, acoustic, olfactory, and trochlear. The facial trunk may be affected in its nucleus or roots, and is said to be affected in the trunk, but this has rarely been seen; the nuclear type is the one generally seen, commonly from softening in the pons from the basilar artery. These paralyses are remarkable in that they are transient, and come and go. The eighth nerve may be degenerated in company with the seventh, or separately. In all cases of cranial nerve palsy, we are apt to see a complicating hemiplegia of the crossed type, since the softenings which affect the nuclei of the cranial nerves are apt also to affect the motor tracts descending to the opposite side of the body. Some cranial nerve palsies are due to the direct effect of pressure, and to infection of a nerve trunk, and therefore do not entail a hemiplegia. On account of softenings about the fourth ventricle at times, polyuria, polydipsia, and glycosuria are developed. These paralyses are the features of the acute stage, which may show improvement, and the general symptoms as well. But the patient generally becomes stuporous, and mentally dulled. He may then go into coma, from which he never recovers. In other cases excitement takes the place of stupor, and he may have wild delirium and convulsions.

In all forms of syphilis of the brain, we encounter mental changes, loss of ethical sense, of mental acuteness, power of concentration, anemia, and dementia of some grade in the terminal stage.

Neurasthenia. The syphilitic patient usually becomes anemic and nervous owing to the physical and mental shock of the infection, and this is often aggravated by the severity of the treatment. A typical neurasthenia often appears during the second stage, sometimes the result of primary arterial changes, but most often caused by meningeal irritation. The sense of pressure upon the head, the vague and indefinite pains and paresthesias, combined with irritability, loss of mental efficiency and insomnia differ in no respect from the symptoms of ordinary neurasthenia. The difference lies in the fact that they have definite pains which may be more severe at night, and the headache may also show an increase in the night. They are also subject to transient hemiplegias, aphasias of slight duration, and sensory disturbances may appear, limited to one side of the body. Pupillary changes may occur, and are always a sign that there is a meningitis of the base, or of the convexity.

Hysteria is quite a common condition, chorea less often, while epilepsy of the classical type is common. All these conditions may be impossible of proper diagnosis without the Wasserman reaction, and an examination of the spinal fluid.

SYPHILITIC DISEASE OF THE PERIPHERAL NERVES. A Neuralgia may be the first symptom which directs the patient's attention to his condition. In any case it is apt to be an early feature of the disease. Trigeminal neuralgia is common, and then that of the cervical plexus, and of the brachial plexus. Intercostal neuralgia is also common, and it may involve the lumbar, and the sacral plexuses. The injury may not be directly to the nerve-trunk, but it is quite commonly due to an inflammation of the perineurium, or epineurium, which finally comprises the nerve-fibres. This is the result of the involvement of the peri-endoneural septum, which conducts the vessels into the nerves, and the congestion of this structure compresses and infects the blood-vessels, causing disturbances of nutrition in the form of atrophy, necrosis, and caseation. Syphilitic root-neuritis is caused by compression from the leptomeninges, and primary nerve involvement combined. There is also the typical form where the nerves are primarily invaded by gumma, and the deposit in the anterior and posterior roots along the whole length of the cord gives the impression of a string of beads.

PERIPHERAL PERINEURITIS. The pathology is similar to that of the root-neuritis, and the same changes take place in the superficial nerves as has been described in the spinal nerves. Gummatous perineuritis has been observed in the ulnar, crural, and peroneal nerves. In perineuritis the pathological changes consist of peri-endoneuritis, gummatosa, arteritis of the vaso-vasorum, simple or degenerative atrophy of the nerve-tissue, and a greater or less degree of caseous necrosis.

SIMPLE SPECIFIC DEGENERATIVE POLYNEURITIS. There is no evidence that there is a specific form. The clinical forms of peripheral nerve syphilis appear as neuralgias.

ERB'S SPINAL SYPHILIS. Spinal syphilis is much more rare than syphilis of the brain, and is only one-fifth as frequent as the cerebro-spinal type. Syphilis of the Spinal cord is only one-tenth as common as tabes. The brain symptoms in the combined form may so overshadow the spinal symptoms that they may be unobserved during life, although very extensive. Gummata may appear in the cord, either multiple or singly, and when they are diffuse they vary in size from a grain of wheat to that of a pea. Softening may occur, either in the white or gray matter, due either to disease of the vessels or to the effect of toxin on the nervous tissue. The gray substance will show cell-degeneration, or hyperemia, or hemorrhage. All parts of the cord may be affected, and patches of softening or sclerosis may extend across the cord, giving the appearance of a transverse myelitis. The white substance is more often affected than the gray, and the posterior rather than the anterior portion of the cord. Where the cells have been destroyed there is a replacement by neuroglia tissue. The roots may be injured by direct compression by the congested membranes, or the inflammation may extend along the roots, or an extension of the arterial inflammation may implicate the vaso-nervorum, or gumma may invade the sheaths of the nerves. The roots are never affected alone, nor are all the fibres ever destroyed. The cord is never primarily affected, but spinal symptoms are usually the result of an interference with the nutrition of the cord as the result of a meningitis. A genuine specific infection never causes a typical system disease. The pathology of spinal syphilis has nothing diagnostically characteristic about it. Oppenheim says that spinal syphilis appears early, one-half the cases occurring in the first six years after infection, but it may, however, be deferred until the twenty-fifth to the thirtieth year after. It is more often seen after severe infections, but the preliminary symptoms may have been slight. Brasch states that the more perfect the preliminary therapy, by so much less is the danger of spinal syphilis, but this is not universally acknowledged. Men are more often victims than are women. Syphilitic diseases of the spinal type take the form of Chronic Spinal Meningitis, Meningomyelitis, Acute Syphilitic Myelitis, Erb's Spinal Syphilis, Gummatous Tumor, and very rarely Syphilitic Diseases of the Vertebræ.

On account of the small size of the cord, and the close proximity of the very important tracts and cell-areas, it is easy to understand the immense damage that a very small lesion can produce. Since the membranes are the usual point of attack, following the infection of the small blood-vessels, and since the folds of the pia insinuate themselves into every part of the cord, and since the blood-vessels follow these folds, it is apparent that every part of the cord is open to attack, and the important role of the arteries is made clear. The membranes of the cord are affected oftener than those of the brain.

ETIOLOGY. Physical exhaustion is a potent factor in inciting the disease. Alcohol, and every other form of excess is efficient, and traumatism often seems to stand in a causative role.

PATHOLOGY. The pathology of spinal syphilis deals with the reaction of the membranes and the cord-substance to the virus, as the vertebræ are very rarely affected, and are scarcely to be considered in the pathology of this disease.

Meningitis may be acute or chronic, localized, or diffused. The acute form almost always originates from the dura, a Pachymeningitis, while the chronic form arises from the arachnoid. In the acute form there is an infiltration of round cells, and an exudation of these, and of connective-tissue cells. There may be patches of gumma, but solitary gumma is rare. This infiltration thickens the membrane, interfering with the whole vascular apparatus, and thus producing congestions and ischemias in the cord, resulting in an irritation of the spine. In addition, the nerve-roots are compressed and irritated. If the process becomes chronic, all the membranes become matted together by the exudate, and there is a fibroid proliferation of the membranes, which may go on to degeneration and caseation. While in the acute form the circulation of the cord is disturbed, and its functions are deranged, in the chronic type the veins, arteries, and lymphatics are so compressed and infected that there are thromboses, not only in the peripheral vessels, but also in those distributed to all parts of the cord. This may result in spots of softening, or if the process is a more gradual one, in sclerosis, not only of the membranes, but also of the cord, so that it feels like a fibrous mass. The affection of the cord in these cases is not primary, but the sclerosis is marginal, or extends along the septa of the pia. The veins are the initial avenue of infection, and the arteries and lymphatics are affected later. When foci of softening or sclerosis are produced, secondary degeneration occurs in the tracts, both ascending and descending.

SYMPTOMATOLOGY. In the beginning of the second stage of syphilitic infection, the signs of meningeal irritation may become evident. There will be backache, paresthesias of the upper and lower extremities, weakness, and a tired feeling in the arms and legs, with slight irritation of the bladder. The skin and tendon reflexes are active. The perception of touch, or pain, or changes of temperature may be disturbed. At this stage Schnabel found a hyperemia in the background of the eye, which he considered to be objective proof that these symptoms were due to a similar hyperemia of the cord. When actual inflammation has taken place, sensory irritation is shown by lancinating pains, which may radiate to both upper and lower limbs, and other parts of the body, but not always disturbed equally, and especially in the lower part of the spine. Owing also to the unequal compression and irritation of the spinal roots, there will be an irregular distribution of the hyperesthesias and vasomotor symptoms. The pains are generally severe, and in some cases are worse at night, and there is always some degree of dysuria. There is a hyper-sensitiveness to touch and pain. The back is stiff, movement of it causes distress, and the vertebræ are tender to percussion. Motor irritation is shown by a feeling of tension in the muscles, which may develope into contraction. The limbs are weaker than usual, but as a rule are not actually paralyzed, although a pseudo-paralysis is feigned at times, from a fear of pain on movement of the limbs. If the meningitis implicates the roots of the last cervical and first dorsal segments, the sympathetic ganglion at that point will be affected. It will produce a retraction and lessening of tension in the globe of the eye, and a narrowing of the palpebral fissure. From the paralysis of this sympathetic ganglion there will be a contraction of the pupil, from a lack of opposition to the action of the third nerve, and stimulation of the skin of the neck and of the cheek will not produce the usual dilatation of the pupil, the oculo-skin reflex. That side of the face will flush and sweat, and will be colder than the other side. The final proof of the specific origin of the meningitis is the discovery of a leucocytosis, and an increase of globulin in the spinal fluid.

The symptoms of cerebro-spinal syphilis are a combination, in a variable degree, of the symptoms of the two types. Basilar meningitis almost always presents some symptoms of spinal meningitis.

MENINGO-MYELITIS. PATHOLOGY. Meningo-myelitis is the most frequent form of spinal cord lesion, and the softening or sclerosis may involve a whole transverse section of the cord, or multiple lesions may be scattered through it. The small blood-vessels of the pia are the point of origin of the round-celled invasion of the membranes, and this infiltration can be traced along all the prolongations of the pia into the cord. The anterior gray matter of the cord, as well as the white substance, is richly supplied with arteries, which radiate along the pial septa into the interior of the cord. The posterior gray matter, as well as the white, is abundantly supplied by its own particular vascular system. In the gray matter the cellular infiltration is at its maximum. In consideration of these facts, and also remembering that it has before been stated that the vascular system is peculiarly vulnerable to the syphilitic virus, it is easy to understand that vascular forms of specific disease of the spinal cord will predominate. Thickening of the vessel-walls contracts their lumen, diminishes the blood-supply, and produces softening of the nervous tissues. Degeneration and atrophy of the nerve-cells, especially of the anterior horn, is sure to follow. In the white matter the fibres first lose their myelin sheaths, and later disintegrate, and the neuroglia proliferates, and replaces them. If the process is chronic, there is a sclerosis instead of softening, which involves both the membranes and the cord, and a section will be found changed into a fibrous mass. The multiple character of the disease must be emphasized as the rule, but not infrequently cases present only one or two symptoms. The meningitis is usually most severe in the dorsal area, since the cord has here the lowest resistance, consequently the myelitis is almost a dorsal one. The fact that the roots, both anterior and posterior, have their own arterial branches, makes possible the developement of root-symptoms through localized arterial disease. Since every focus of softening or sclerosis necessitates secondary degeneration, the symptomatology is based upon degeneration of tracts and cell-areas, and on account of its erratic distribution, we may find the incomplete types of myelitis producing symptoms of disease of the anterior and lateral, or of the posterior columns.

SYMPTOMATOLOGY. In the majority of cases the meninges are affected days or weeks before the myelitis occurs. The symptoms are the same as those from a meningitis and a myelitis from other causes, with the exception that possibly the pains may be more pronounced at night. This is a classical statement, but Nonne says that literature fails to substantiate it. The inflammation of the meninges, and the irritation of the roots give rise to pains in the back, and between the shoulders, which may radiate to the extremities, and from the legs to the feet, if the cauda equina is affected. There are numbness and tingling in the limbs at the same time, which develope into a hyperesthesia, and a diffuse sensitiveness to pressure in the painful areas, tenderness of the spinal column to percussion, and painfulness on movement. The motor irritation is shown by rigidity of the spinal muscles, and there may be paralyses of muscles, with atrophy and electrical changes, from a deeper involvement of the spinal roots. Hyperesthesias in spots, vasomotor paralyses, and slight dysuria, and vesical irritation, are also seen to some degree at all times. There may be a slight paresis of the limbs, also, as a meningitic symptom. All of these symptoms are transient, and frequently change their localization. The skin and tendon reflexes are increased. Kernig's sign is present. When the cord becomes involved paralysis developes in one or both the extremities, particularly in the lower, and usually more in one than in the other. It is flaccid in the beginning, but later becomes spastic. The skin reflexes may be increased, or diminished, while the tendon reflexes are usually increased, and Babinski's and Oppenheim's reflexes, one or both, are present. The sensations are disturbed, hyperesthesia or anesthesia may occur, as well as dissociation of all kinds, reminding one of syringomyelia, or less often of the Brown-Sequard syndrome. At times the perception of cold may be lost, and that of heat be lost, or the opposite. The sphincters are affected early, and universally, early in the form of dysuria, and later as incontinence. Sexual power is impaired or lost. Atrophies appear in various muscle-groups, and the impaired nutrition and function of the bladder make it the frequent focus of a fatal infection. Bedsores are a frequent incident, and are a source of exhaustion and infection. The symptoms in these cases are characteristically diffused, bilateral, and transient. According to the location of the spinal lesions the disease may assume the form of almost any disease of the cord. When in spite of treatment, it advances, the prognosis is the same as in any case of chronic myelitis. If the patient recovers, the weakness of the bladder is liable to persist, and there are weakness of gait, and stiffness of the spine, and some losses of functional ability in various directions.

LUMBAR MYELITIS. This is a less frequent form. When a transverse lesion does develope, there will be a flaccid paralysis, with a loss of the skin and tendon reflexes, and the sensations will be perverted, as in a dorsal myelitis. The disturbance of the bladder may be a weakness of the viscus, or of the sphincter, or a mixture of the two, ischuria paradoxica. These cases are especially liable to bedsores, and severe disturbances of nutrition. Frequently the tendon reflexes of the upper extremities are exaggerated, indicating that the meninges are hyperemic.

CERVICAL MYELITIS. In these cases all four extremities undergo motor and sensory disturbances. Sensation is rather selectively affected. If the 8th cervical, and the 1st dorsal are involved, the sympathetic ganglion is affected, the globe of the eye is retracted, it is smaller, the pupil is dilated, the palpebral fissure is narrowed, that side of the face shows anomalies of sweat secretion, and is colder than is normal. Symptoms may occur singly, or a group of symptoms may appear simultaneously, and in many cases we shall find brain symptoms preceding the cord symptoms. The patient may suffer for a time with severe headache, or attacks of vertigo, or transient attacks of aphasia of some degree. He may have paresthesias upon one side, or the limbs may be weakened. In all forms of meningo-myelitis the symptoms are very variable in distribution and severity.

COURSE. Meningo-myelitis is almost always a chronic disease, but occasionally it sets in acutely. In such cases, after a few symptoms of meningitis there is a paresis of one or more extremities. The disease tends to progress uninterruptedly, but some cases show remissions for months or longer, and then resume progress toward a fatal issue. Some cases regress to a practical recovery under treatment, but this improvement may be unilateral, and relate only to sensation, or to motion. In any event, recovery is almost certain to leave the patient partially defective in one or several of the functional activities. At any stage a chronic meningo-myelitis may develope into an acute myelitis. A small per cent. of cases acutely progress to a fatal issue, death resulting from bedsores, or cystitis alone, or in combination with pyelonephritis.

TREATMENT. The only efficient treatment for these cases is an antisyphilitic therapy, Salvarsan, Mercury, and the Iodides. It is probable that the intraspinal method of treatment will prove to be the most efficient, and possibly the only efficient method of application of these remedies in severe cases.

ERB'S SYPHILITIC SPINAL PARALYSIS. The most common form of spinal cord syphilis is a complete transverse dorsal myelitis, which Erb, in 1892, defined as follows: several years after a syphilitic infection, a slow and gradual developement of a spastic paresis of the lower extremities apears, with spastic gait, motor paralysis, slight muscle tension, slight increase of the tendon reflexes, sensory disturbances, either slight, severe, or absent, and bladder weakness. The upper extremities, as well as the intelligence, the cranial nerves, and the pupils remain normal. Existence of this syndrome has been doubted, but cases occur which conform very definitely to this description.

SIMPLE ACUTE MYELITIS. After infection, or in the early part of the secondary stage, attacks of acute transverse myelitis have been reported.

These cases cannot be pathologically separated from the myelitis of the ordinary type from softening.

ACUTE SPECIFIC TRANSVERSE MYELITIS. This form of myelitis is relatively frequent compared with the non-syphilitic forms, and there is a variation from it in the degree of paralysis, both of sensation and of motion. It usually appears in a syphilitic who is still able to go about, although he may be limited in his ability to work. It may come within a few hours upon an individual while in apparent health, but promonitory signs usually precede the appearance of the paralysis. Rosin claims that the prodromal stage may be predicted by the peculiarities of the reaction of the patellar reflex. At different times it will show exaggeration, to be followed by a period of normal reaction, which later may change to diminution. This irregularity is a phase which may be recognized, not only for days and weeks before the onset of the myelitis, but even for months and years. The premonitory symptoms are, on the sensory side, paresthesias, radiating, shooting, boring pains in the lower extremities, hips, parts of the trunk, and spinal column. Charcot and Oppenheim both have said that the stiffness of the back, and sensitiveness of the trunk to movement was indicative of spinal irritation, and was characteristic and common. The motor symptoms are irritative, and are twitchings, and spasm-like movements of the toes, feet, and legs, which alternate with the actual paretic condition. Very often disturbances of the bladder and rectum, in the form of retention, precede the occurrence of the transverse paralysis. Next paralysis appears, perhaps in a few hours, or in a few days, and affects the lower extremities. This is almost always an absolute paralysis below the pelvis. While the upper extremities may be involved in the paralysis, it is unusual. Sensation is usually severely affected, but it may be in an irregular manner, and the Brown-Sequard syndrome may be present. The sphincters of the bladder and rectum are severely compromised from the first, but the tendon reflexes will show a variability of change both in localization and degree. In many cases edema quickly makes its appearance in the paralyzed parts, and bedsores develope in the sacral region.

COURSE. The disease usually runs a rapid and fatal course, but remissions may occur. Examination shows that both the anterior and posterior spinal arteries are affected with a severe arteritis of the Huebner type. General inflammatory symptoms are absent, as is gumma, but there is an inflammation of the pia, with softening in the cord. There is an incongruity between the pathological findings, and the clinical symptoms. This is explained by the variable involvement of the arteries to the motor and sensory areas of the cord. At the beginning there may be a meningitis, but thrombosis does not always follow it.

GUMMA. Gumma usually arises from the meninges, and is generally multiple. Its symptoms differ in no respect from those of the ordinary tumors of the spine. It is not very suitable for relief by operation, but does respond readily to remedies.

CEREBRO-SPINAL SYPHILIS gives rise to a combination picture made up from the foregoing descriptions of cerebral and spinal syphilis. It is a more common manifestation than that of either location separately.

PERIPHERAL NERVE DEGENERATION as the result of a definite action upon the nerve structures is rare, and denied by many. Some claim that it specifically affects the circulation of the nerve, and so induces degeneration, but it is more probable that except in rare cases the nerve degenerates from softening involving the nucleus, or by the compression of a nerve trunk by a gumma, or infiltration into the sheath by the same, or the degeneration results from the compression of the roots by the thickening of the meninges.

TREATMENT. In view of the kind of symptomatology which we derive from a study of this class of cases, symptomatic prescriptions are apt to be disappointing, while physiological remedies are often brilliantly successful. It is therefore my opinion that treatment by Salvarsan and the Mercurials should be made use of. They are not always successful, and in such cases Homœopathic medication may be useful. Suggestions for such treatment may be found in the articles on Myelitis, Meningitis, and Tabes.

CHAPTER XIV.

FUNCTIONAL NERVOUS DISEASES.

EPILEPSY.

Idiopathic Epilepsy, Falling Sickness, Morbus Sacer.

Definition. Satisfactory definition is difficult, since actual pathognomonic signs are rare. Nevertheless it may be fairly stated thus: It is a recurrent paroxysmal neurosis, in which some irritant acting upon a neuropathic constitution induces attacks which disturb consciousness, or produce motor incoördination, or commonly both at the same time. The attack may or may not be marked by convulsions, and a Major, Minor, Focal, or Psychic type, are recognized.

The Symptoms of a Typical Case. A typical Major or Grand Mal attack is a sudden brief recurrent discharge of nervous energy, not due to a normal stimulus for such discharge. The person will at intervals be prostrated by a convulsion which will begin in a tonic form, and end in a clonic spasm. It will begin in a part, and finally involve the whole body in the spasm. During this convulsion, foam will be ejected from the mouth, the sphincters will be relaxed, the tongue will be bitten, and consciousness will be lost. The attack will last from one to three minutes, when the person will fall into a deep sleep, from which he will awaken somewhat confused, and mentally and physically exhausted.

Differential Diagnosis. To be differentiated from Focal or Jacksonian Epilepsy, Intoxications, Hysteria, Migraine, and Psychogenetic Convulsions.

Race. It is confined to no climate or latitude, or racial division of the earth's population.

Sex. Males predominate in the proportion of 20 males to 16 females. In the first 10 years males predominate, in the second 10 years there are more women. In 913 cases 59.2% were male. (Siebold.)

Age. It may attack the child in infancy, and has been known to occur at the seventy-fifth year, but after the age of thirty it usually results from traumatism producing organic lesions, from arteriosclerosis, syphilis, and the various intoxications, of which alcohol is the most common. The fourteenth year is the point of greatest incidence, 25% begin between the birth and the sixth year, 54% between ten and twenty-two or three (Turner). In 913 cases, 83% were attacked before the twentieth year (Seibold).

HEREDITY. Direct heredity is more common than in any other cerebral disorder. Echeverria, out of 533 cases, found direct inheritance in 29.72%; Gowers found it in 35%; Spratling in 66%; Seibold out of 913 cases 34.8%. Those with heredity take it earlier than those without. (Seibold.)

ETIOLOGY. Epilepsy may be directly inherited from an ancestor, and hence epileptics should not marry. The inheritance may be indirect, in that the parents may endow their offspring with an instability of the cortical or subcortical cells, which renders them unable to withstand the effects of endogenous or exogenous toxins, traumatism, or the strain of physiological developement, or the demands of competitive existence. Alcoholism in a parent is one of the most common causes of epilepsy in the child, and if both parents are addicted to its use, such a result is almost certain; parental alcoholism has been estimated to cause from 10% to 18% of all cases. The latter percentage is from Seibold, and he states that parental indulgence is many times more potent than that of the individual himself. Parental intoxication at the time of conception is very disastrous, and this is proven by a curious wedding custom in a certain country in Central Europe; there it is customary to follow a wedding with a carouse, only terminating with the endurance of the participants, and it has been noticed that a considerable percentage of the first children of such families are epileptics. Much of the instability of nervous tissue results from intrauterine disease, resulting from the acute infection of the mother during gestation. Epilepsy in a child of from five to seven years should cause us to look for syphilis. Spratling considers that epilepsy developing before the tenth year is probably from bad heredity, cerebral palsy, or is the sequel of an infectious fever. In the period from the twelfth to the sixteenth year inclusive, it is the result of the changes of puberty; this is the particular age for the developement of essential idiopathic epilepsy (the fourteenth year is the exact point), but is not so efficient as an exciting cause in males as in females. Fright has been considered to be the efficient cause in about 5% of the cases.

With a practical agreement that toxins were the exciting causes of the attacks, immediately arose the natural question, "What is the toxin?" Guido has shown experimentally that there are profound metabolic abnormalities in the epileptic; they are not as normal individuals. The most manifest deviation is in the production of urea; hence an acid intoxication, and an excess of ammoniacal compounds is found in the urine of epileptics. Removal of these chemical compounds by an appropriate diet reduces the number and intensity of the seizures. Donath found cholin in the cerebrospinal fluid, and that it was an active hemolytic. Gelma states that thyroid gland changes are frequent, but not universal, in epileptics. Others have assumed that the attacks were due to aberrations in the secretions of this gland, either a pathological increase or decrease, and affecting the cortical cells directly, or that the toxins arising from the normal metabolism of the body were not neutralized, as they are normally, by the action of the thyroid and parathyroid. Some color to this hypothesis is given by the statement of Myers that persons have developed true epilepsy from overdosage with thyroid substance, and from the report of Britton that he has successfully treated forty patients, reducing the frequency of the attacks, or stopping them altogether, by feeding them the expressed juice of beef thyroids.

The so-called reflex causes are only irritants to a neuropathic constitution, and are not in themselves competent to inaugurate an epilepsy in a healthy person. Tucker, Comby, and others have insisted that birth traumatism is a great factor in the causation of epilepsy, and have urged more frequent and prompt surgical interference. Spratling says the old lesions of an infantile cerebral hemiplegia are the most frequent found in the brains of epileptics. The percentage of diplegias and hemiplegias that afterward become epileptic was 44.3%, Sachs and Peterson; 26%, Osler; Wallenberg, 66 in 160 cases; and Lovett reports 26 severe cases of which 96% became epileptic. If a person, at any stage of life, suffers an injury to the skull which either irritates or compresses the cortex, a focal epilepsy is likely to result (10%, Seibold). Denk, after considerable experimental work on animals, analyses 33 cases of brain tumor, only 5 of which were without epilepsy, and 20 cases of epilepsy, with operation or post-mortem findings in nearly all, and comes to this conclusion: aseptic organic changes in the brain are not of themselves competent to produce epilepsy. An accessory factor seems necessary. If we presuppose a hereditary weakness in the cortical cells, from alcoholism, insanity, or chronic intoxication in an ancestor after such an injury as has just been described, after a variable period of time there may arise a generalized epilepsy, differing in no particular from the epilepsy arising from other causes. Senile epilepsies differ from the ordinary type in having their origin in claudication of the arteries of the brain, or in failure of power in the heart, resulting in a blood-pressure either too low or too high for orderly functions in the brain. Russell reports an interesting case of epilepsy occurring in a man aged twenty-one years, in which, during the onset of the spasm the pulse disappeared, remaining absent for three-quarters of a minute. Sudden cerebral anemia, due to cardiac inhibition (or to cerebral vasomotor spasm) would account for most of the symptoms of an epileptic fit; the recovery from the fits may find adequate explanation in the vagus escape of the heart. Certain morbid changes have been described in the brains of epileptics, but it is possible that they may be secondary to the repeated attacks of transient cerebral anemia, which must follow cardiac arrest, and to the extreme congestion of the brain which the convulsive movements give rise to.

PATHOLOGY. It has been definitely decided that the irritation of the cortical cells gives rise to the clonic form of convulsion, while the subcortical (Samaja says "at the base," while others limit it to the pons,) are responsible for the tonic form. Southard says that hereditary cases show during life, or at autopsy, signs of organic nervous disease, while the acquired cases usually display an original focus, such as an infarction, hemorrhage, or tumor, which incited the epileptic seizures. In nine out of ten cases they were found in the motor area, either in the occipital lobe, or in the hippocampal gyrus. He also found that there was a Marchi degeneration of a non-systematic type in every epileptic cord examined. Turner says that pronounced changes may be discovered in the brains of epileptics, but that the frequent repetition of epileptic seizures brings about structural changes in the nerve-cells and fibres whose place is taken by proliferated neuroglia elements. Finally, examination of the results of 3,000 autopsies revealed a large percentage of brains which were undersized or asymmetrical, or showed lesions in various localities, perhaps preponderatingly in the cornu ammonis and the medulla, but with no such unanimity between convulsive seizures and any definite lesion as would be necessary, if one were to consider justly that they stood in the relation of cause and effect. All these considerations have led to a practical agreement that the pathological change is in the cortical cells. Cortical, because it has been noticed that if an epileptic suffered from a cerebral hemorrhage which injured the internal capsule of one side, the paralyzed side was no longer a participant in the convulsions which were formerly general in their distribution. Next, that the pathological condition of these cells is an hereditary or acquired weakness which is not gross enough to be visible. There is some ground for belief that the second, or inhibitory layer is especially affected. Granting this cellular weakness, it is believed that traumatism, infection, intoxications, both endogenous and exogenous, may so act upon this debilitated second cortical layer that the motor cells of the third layer, lacking the former inhibitory restraint, respond, after a period of rest, to peripheral stimuli in irregular and disproportionate motor spasms, until the period of exhaustion supervenes. This would explain the frequency of attacks after gluttony, obstinate constipation, and aberration of glandular secretion. It is a somewhat significant fact that convulsions of an epileptic character with an aura of a peculiar smell can be produced by the pressure of a tumor on the uncinate gyrus. Psychical epilepsy has been produced in this manner.

Symptomatology. The attack may be without premonitory symptoms, but often the patient will for some hours or days display irritability, or conversely, be apathetic, and show motor retardation. The author found in a considerable group of epileptic dements a premonitory increase in indican. Onuf and Lagrasso found in the urine of epileptics a great fluctuation in the leucocyte count. When intervals between the attacks were long, the fluctuations were apt to be slight; a rise seemed to predict a seizure, and the deduction seemed to be that a meningo-encephalitis was the cause of the attack. Stadelmann refers to a phase of naughtiness, moodiness, and brutality in children, with a tripping of their words in their haste to speak, as premonitory signs. Immediately preceding the attack, a large majority of cases (Spratling says 50%) are apprised of its onset by a definite phenomenon called an aura, and this is practically always the same for the individual. Auræ are psychic, sensory, motor, and irregular. The psychic aura is often an abnormal mental state, of which the most common is an acceleration of the imagination, confusion of ideas, and fear. This latter is the most common of all. The sensory auræ have a wide range, of which flashes of light are the most frequent. Optical illusions are sometimes experienced; one patient always saw a green woman, who struck him, and then consciousness departed. Some patients become blind, but a dazling play of colors may appear, of which red and blue are the most common. Auditory auræ may exist alone, like the sound of waves, or a buzzing noise, but are often combined with the visual disturbances. Much more rarely olfactory auræ (smells bad, or only peculiar) may precede the attack. Epigastric sensations bulk more largely than all others combined, and are often the source of the aura of fear. A heat sensation rising from the stomach until it chokes the sufferer, is one form. Vertigo is another aura of great frequency. Another common sensory aura is a numbness creeping up a limb. Motor auræ are usually a run of a few steps, or turning movements of a part, or of the whole body. Alfred Gordon reports the case of a boy of twelve, suffering with essential epilepsy since his second year, who, for about forty-eight hours before an attack, was constantly going about kissing everybody he met, the kissing being sometimes replaced by laughing, and sometimes by slapping or striking. During all this time he was apparently conscious, and realized what he was about. After forty hours or more of this, he would have the epileptic attack. When a large aggregate of cases are observed, the great variation in symptomatology is such a striking fact that the description of a clear cut characteristic syndrome has to be abandoned, but the majority of cases present enough symptoms in common to justify the attempt to delineate a classical case. After a possible period of malaise, the patient experiences the aura, utters a low tremulous groan, runs a few steps, may clutch a near-by person or object, and *falls to the ground with no regard to his personal danger or appearance.* The face now becomes pale, the eyes are turned upward; pupils are usually dilated, but may be contracted, or show no change. The body becomes rigid in the stage of *tonic* convulsion, the arms are held away from the body, with the forearms flexed upon the upper arms, the fingers strongly contracted with the thumbs turned inward on the palm, while the legs are strongly extended. This stage lasts about twenty seconds. Now the picture suddenly changes, and the body and the limbs are jerked violently to and fro in *clonic* convulsion. These jactitations may be so violent as to throw a person from a bed, and have often produced dislocations of bones, the shoulder joint suffering most frequently. The muscles of the jaws, and of the mouth and throat share in this rhythmical jerking, with the result that the saliva wells out of the mouth, streaked with blood from the lacerated tongue. The sphincter of the bladder often gives way before the spasm of the abdominal muscles, and the urine is voided in spurts, and, less often, there is incontinence of the bowels also. In this clonic stage the pale face becomes congested and bloated, and hemorrhages often occur in the conjunctiva, and less often upon the cheeks. The complete tonic and clonic cycle lasts from one to two minutes, after which the patient falls into a deep sleep of from thirty minutes to two or three hours, from which he awakens mentally and physically exhausted, but generally very hungry, and totally unconscious of having had the attack. For this reason nocturnal attacks in an unobservant household may exist undiagnosed for a long time. Asymmetry of spasm is quite common. At the onset of the attack the knee-jerk is abolished; for the next few moments it it exaggerated, and it is then abolished for a day or two. Ankle-clonus may be present for a time after the attack. In general it must be said that the state of the reflexes shows great variation. Many cases are nocturnal, or are so for a long time, but finally become indifferent to time. In the inception of the disease there is a considerable variation in the frequency of the attacks, but they are apt finally to assume a rather definite periodicity. When they arise from pre-natal injury or marked hereditary taint, and become manifest in the first two years of life, they are apt to be repeated at intervals of a week to a month or two for a year, or thereabouts. They then disappear until they reappear at puberty, or late adolescence. They then take on the type of cases originating at that age; and at first occur only three or four times a year, but grow more and more frequent, until they establish their own peculiar periodicity. Spratling says that out of 1,374 cases of all grades of severity the following frequency was established: weekly, or oftener, 5%; every two weeks, 13%; every three weeks, 4%; every four weeks, 18%; every six weeks, 2%; every eight weeks, 3%; every twelve weeks, 4%. The large increase at the monthly interval corresponds to the expected result of menstruation.

Gowers gives three characteristic features to that form of epilepsy resulting from organic diseases of the brain during infancy. 1st, The epileptic convulsions are more or less one-sided; 2nd, first attacks of infantile convulsions are of great severity; 3rd, first convulsions often occur during some acute illness, soon after a fall, or in a state of general physical prostration.

Minor or Petit Mal attacks are found alone, or in combination with the Major or Grand Mal attacks just described. These Petit Mal attacks consist of attacks of momentary unconsciousness, forced movements of various kinds, either of the whole body, or some motor function, or the ejaculation of some word or phrase. It may be only a momentary halt in conversation, or on the other hand the utterance of a disconcerted exclamation. They are liable to occur very frequently in a single day. If occurring alone they are prognostic of future Grand Mal seizures.

Psychic Attacks, or Psychic Equivalents. At this place it should be pointed out that after an epileptic convulsion of the major type there are often temporary paralysis, and various forms of delirium. These conditions may so closely simulate gross lesions of the brain that operation has been undertaken for the relief of a condition, which the lapse of a day or two in time would have entirely resolved. The true psychic equivalents or crises are certain psychic states which to some degree take the place of epileptic seizures. They must be differentiated from Psychogenetic attacks which are not epileptic in any way but are pure psychoses. When a patient suffers from obsessions, impulses, manias of interrogation, tics, phobias of various sorts, he must be considered to labor under a profound disturbance of the feelings, the will, and perception. Developing slowly it becomes a psychesthenia; appearing stormily, and reaching a climax rapidly, so that when a veritable crisis is attained it will be a psycho-epileptic crisis or equivalent. These attacks may be momentary, like the motor attacks, or they may last weeks and months, and take the form of bizarre actions, or breaches of manners, or morals, the commission of a crime, or the undertaking of long journeys, and the complete change of one's means of livelihood. They may be inaugurated, or terminated, by convulsion, by an injury, or illness, or be entered upon, or ended, without any outward influence or manifestation. A certain man may leave his work, and invade the privacy of utter strangers; he may start from his home with a cry, and run through the fields in a great circuit; or change his habitation, and entire mode of life. A patient of the author has several times deserted, to its ruin, a very profitable business.

The interparoxysmal mental condition of epileptics is not wholly the result of the seizures, but is an expression of the same degenerated constitution that gives the basis for the attacks. Epileptics tend to dementia of a paranoid or catatonic type. The paranoid patient has systematized delusions, ideas of persecution, loss of all sentiments of affection, convulsive paroxysms and states of violence, particularly at the menstrual period. The catatonic type is marked by catatonic stupor, suggestibility, verbigeration, and negativism, which may go so far that the patient may have to be fed forcibly. Other psychic aberrations are emotional states with changes of temper, usually melancholic, but may be maniacal. They may have hallucinations, stupor, and delirium, usually with sensory illusions, and a marked tendency toward frenzy. Dream states are also seen.

Status Epilepticus. Sometimes one convulsion does not terminate the attack, but they recur at short intervals for days. This is termed the "Status Epilepticus," and often proves fatal. Epileptic status in pregnancy is easily mistaken for eclampsia, and error in diagnosis is a serious matter, since the treatment is quite different. Eclampsia is diagnosed by the low freezing point of the urine, and the higher grade of the hyperleucocytosis.

PROGNOSIS. It is not a self-limited disease, and opinion has been inclined toward considering it almost incurable. This has been the result of the impression made upon literature by those who believe in epilepsy as an entity, and in bromides as the only treatment. The latest investigations have emphasized the fact, that it is not epilepsy, but one of the epilepsies that we are considering, and consequently that the management, hygiene and medication must vary with the type of the disease. Turner says that the discovery of the bromides has not bettered the prognosis, and Spratling says that he has yet to learn of a single case of epilepsy cured by the bromides alone. At present we may consider, that, even in chronic cases, five per cent. of cures may be expected, and taking cases together, both recent and chronic, the percentage of cure is held to be from 10 to 25%. Sex has nothing to do with this probability, nor has the history of inheritance; but epilepsy beginning in infancy or childhood, or when its approach is delayed until adult life, has an unfavorable prognosis. The epilepsy of old age is quite amenable to treatment, while that beginning at puberty responds most readily of all. The earlier a case is brought under treatment, and the longer the interval between attacks, by so much is the probability of cure increased. Major attacks are more amenable to drugs than minor, and nocturnal than those occurring during the day. Sometimes a remission which promises cure may be terminated by an accident, childbirth, or an inflammatory disease. It is not true that the changes of puberty tend to cure an existent epilepsy, for the contrary is apt to be the fact, nor should marriage be advised as a therapeutic measure, for pregnancy may light up an epilepsy which has been quiescent since early childhood, but on the other hand, it has cured a few cases. No case should be expected to improve unless the physician is assured of continuity of treatment for from two to three years. If improvement is to result from treatment, it is usually manifested within a year from its inauguration. The mental impairment which so often results, may, with some probability, be attributed to the effect of the immoderate use of drugs; however that may be, mental impairment is more likely to follow minor attacks than the major, but a combination of the two most surely provokes it. Females are most likely to develope epileptic dementia. A few authorities stoutly deny the optimism expressed in this section.

DIAGNOSIS. If it were demanded that a picture should be framed to which all cases should conform, diagnosis would be impossible, since there is no one symptom which is absolutely pathognomonic of epilepsy, but upon the positive side a definite statement can be made with a few chances of error. The case is one of idiopathic epilepsy if a person, after uttering a sharp cry or groan, runs a few steps, or grasps some near-by person or object, and then, with a pale face, has a tonic convulsion for twenty or thirty seconds, falling, not sliding, to the ground, then for a minute or so is thrown into violent clonic convulsions, during which the face is congested, the tongue is bitten, bloody froth is extruded from the mouth, the urine, and perhaps the feces, are voided unconsciously, and the clonic stage is terminated by convulsive grunting. All this time the patient is unconscious, and after the attack falls into a deep sleep. The tonic, preceding the clonic stage is diagnostic, if we see the patient early enough to observe it; the fact that the patient bites the tongue, and in the fall, and subsequent convulsion, frequently injures himself is strong corroborative evidence. The close relation between mental disability, and facial expression is nowhere seen more graphically than in cases of confirmed epilepsy, and the most common example is facial asymmetry or deformity. Clark, Scripture, and Pierce claim that a diagnostic change takes place in the voice, and have charted it. While in a normal person every vowel has a rising and a falling melody, the epileptic runs them along on even tones, and this has no parallel in any other nervous disorder. The patient cannot be taught to sing melodiously. There is a negligent monotony of speech differing from the tired monotony of the neurasthenic, or the strained monotony of the stutterer.

Differential Diagnosis. Organic Brain Disease may produce convulsions of a general character, but they are more severe, tend to last much longer than a minute or two, which is the invariable rule of epilepsy, and they recur in groups far more frequently than is true of epilepsy.

Focal or Jacksonian Epilepsy differs from the Idiopathic form principally in these particulars, i. e., that the former has the clonic preceding, or alternating with the tonic form of convulsion; the focal convulsion is also longer in duration, and is less apt to be general in its distribution. The convulsions are often as frequent as one or more daily, during the first year of their establishment, while such a frequency is never seen initially in the idiopathic variety. It must, however, be recognized that a Jacksonian epilepsy may be simply an integral part of the entire expression of a case of idiopathic epilepsy.

Intoxications may develope generalized convulsions with quite a similarity of symptomatology but here again the frequency is greater, the sequence of symptoms is not so regular for the given case, and while the epileptic is quite apt, especially in the early stages, to be in robust health, the sufferer from an intoxication sufficient to produce convulsions is the subject of a cachexia.

Hysteria is such a protean disease that there may be a close resemblance; in fact, it sometimes can be differentiated only by careful psychoanalytic study. It has been said that a differential diagnosis can be made between epilepsy and hysteria by observing the pupils, which in the former are wide and reactionless, while in the latter affection their reaction is preserved.

Examination will usually reveal definite points. In the hysterical convulsion the movements are wider and more purposeful, and yet, withal, the patient does not expose himself to injury, either when he falls, or when he throws his limbs about in the convulsion. The attack is not marked by the same degree of unconsciousness, nor is it so uniformly terminated by a deep sleep. The tongue is not bitten, although the lips may be; the eyes are not fixed, but are in constant motion, and the lids flutter; the knee-jerks are exaggerated, while the plantar reflex is lost, and the sphincters are not relaxed. The termination of the attack is often marked by a profuse passage of colorless urine. On examination of the urine, it will be found that in hysteria the urea, phosphates, and ethereal sulphates are reduced greatly, and albumin may appear. It is not uncommon for an epileptic attack to be followed by an hysterical seizure.

Apoplexy may be suggested to one when the patient is found in the comatose condition, but epilepsy is a disease of youth, with no signs of arterial degeneration; the face at this stage of the attack has lost its congested appearance; the coma is not so deep but that the patient can be aroused; the pupils are generally even; there is no sign of paralysis when aroused. Positively, we find that the tongue, head, or limbs of the epileptic show scars from former attacks, and hemorrhages on the conjunctiva and the cheeks are common. A delay of an hour or two will terminate the coma of an epileptic, while it still persists in the case of the apoplectic. A history of former attacks is generally obtainable in the epileptic.

Migraine. This is sometimes much nearer epilepsy in symptomatology than would at first occur to one. Gowers has pointed out that the two may co-exist but in such cases the migraine is seldom hemicranial unless the epilepsy is of the minor type. The characteristic prodrome, or aura, of migraine is visual, and when so is characteristically unlike that of epilepsy. The most frequent form is that of a small star near the fixing-point; it enlarges at one side, its rays expanding in zigzags which are often colored,—the so-called "fortification spectrum"—within which vision is dimmed. It lasts a quarter of an hour, or more, and then the headache comes on, usually on the opposite side of the head. Another premonitory sign is "pins and needles" in the hands, slowly ascending the arm, leaving behind it numbness and diminished sensibility. This lasts a quarter of an hour also, and is then followed by headache on the opposite side of the head. The aura of epilepsy only lasts a few seconds and the aura of migraine never begins in the foot. When the right arm is affected there may be a transient aphasia in both affections. The difference between the two conditions is definite and distinctive. Severe pain of long duration does not follow epilepsy, nor is it one-sided. Vomiting in epilepsy occurs soon after the fit and not after the headache has lasted some hours, as in migraine. Bromide is diagnostically efficient in epilepsy; nitroglycerine combined with strychnine in migraine.

Psychogenetic Convulsions. It must be recognized, since psychoanalysis has revealed the large role played by psychic trauma in the etiology of many obscure mental conditions, that certain psychoses simulate conditions which have been supposed to have long since been labeled and catalogued for all time as pathological conditions with a material, pathological basis. It must still be recognized that there may be such a state as psychic epilepsy, but there is certainly a psychosis which may very properly be termed Psychogenetic convulsions. These convulsions are with great difficulty distinguished from true epilepsy since there may be an aura, the patient may fall and injure himself, and in exceptional cases it has been proved to have been a true psychosis although the patient may have bitten his tongue, have had incontinence of the bladder and has lost consciousness. It is not advisable to term such an attack epilepsy, unless there is a family history of similar or allied conditions interchangeable with forms of motor epilepsy, or association in the same person with such epileptic manifestations, the existence of definite epileptic characteristics, such as sudden onset, transitory duration, irregular periodicity, or some of the recognized stigmata of the disease, i. e., change in the character, voice, disposition or intelligence. I have seen such convulsions follow the sudden shock of arrest by detectives.

TREATMENT. If the aura gives ample warning, the patient may ward off an attack by inhaling Amyl nitrite, a glass ampule of which may be carried in a handkerchief, and crushed between the hands, at the first intimation of an attack, or if in a limb, keeping on it a strap with which the limb may be quickly compressed will in some cases stop the attack at that point. An epileptic seizure cannot be stopped if it is well inaugurated, and our efforts should then be directed toward the protection of the patient. No matter what the nature or cause of the convulsion, the victim's clothing should be loosened, and, if possible, he should be placed on a couch or bed pushed up against the wall, and protected on the outer side by cushioned chairs, or vigilant attendants. Put a cork, or toothbrush handle, wrapped with a cloth, between the teeth, so as to prevent the biting of the tongue.

The Homœopathic School has always individualized; it has never treated a pathological condition, but a definite organism disturbed by a pathological condition. It is gratifying now to hear from all sides that there is no one drug of universal applicability to all cases of epilepsy, and that we must treat the patient with the disease, and not the disease. Agreement has been reached that there is a condition of hyper-susceptibility of brain cells, and that the irritant is some toxin. It is, therefore, evident that hygiene is a large element in the treatment, and that it is basic. With poor hygiene no remedy is efficient, and with good hygiene very little medicine will produce a large measure of relief. The patient should sleep in a cool and well ventilated room, should live out of doors as much as possible, and should exercise every day to a point just short of the fatigue that will not be abolished by a night's sleep. Fresh air is as important in the treatment of epilepsy as it is in the treatment of tuberculosis. Sleep should be abundant, never less than eight to ten hours, and in the early part of the night. Women are the better for performing the household tasks that are always at hand. The circulation of the epileptic is always poor, and we should, therefore, enjoin cold baths, spinal douches and massage, and when sleeping the extremities should always be kept warm. Epileptics usually overeat, and often bolt their food. They should be taught to eat slowly, masticate well, drink plenty of water, reduce nitrogenous food to a minimum, making fish and milk take the place of meat, eat sparingly of peas, and beans, and be moderate in the use of oatmeal among the cereals. The bowels must move regularly, and while, for temporary needs, cathartics are not an abomination in my sight, I much prefer to administer two tablespoonfuls of wheat bran in cold water, or in any other vehicle, morning and night, since it is efficient, and tends to permanently cure the condition, and the patient does not need more and more, and finally different drugs, as is the case with cathartics.

Dechlorinization has been a fetich of late years, ranging all the way from a fifty per cent. reduction in the salt, both on the table and in cookery, to a complete replacement of sodium chloride by the salts of bromine. It is generally agreed that patients may have to be forcibly fed after a short time, and some have said that it is impossible to continue the plan more than four to six weeks, not only on account of its physical results on the nutrition of the patient, but also on account of the pronounced mental reduction. The sole reason for its employment is the fact that it about doubles the efficiency of the bromides. If one does not give the bromides, its applicability is not evident. Ulrich is a dissenter. He says that the administration of salt removes the fetor of the breath, and in a 10% solution is an application which will cure the bromide acne. He also uses it diagnostically, and says that the administration of 30 grammes of salt for two successive mornings will bring to the surface a latent epilepsy. If the patient is anemic, use cod-liver oil, but never alcohol, and it is to be kept in mind that most of the concentrated and specialized foods are highly alcoholic.

Reflex irritation has always been a favorite etiology with those who have never seen any large number of epileptics under conditions favorable to judicial conclusions. When an unstable condition of the nervous system exists, it is advisable to remove all evident pathological conditions which would deplete the vitality of the person. Bad condition of the teeth should be corrected, since oral disease has a definite influence on digestion; diseased conditions of the eyes, nose, and orifices and organs of the body may very properly be corrected, but cases are rare where such operations have resulted in a lasting cure of epilepsy. Spratling says that he has never seen the correction of visual defects by glasses produce any lasting effect, neither has the author, but he has seen a cure result from the correction of muscular imbalance. Jacobi states that circumcision is futile. Gowers says that operations upon the generative organs have no beneficial effect. No uterine or ovarian disease can be considered a cause of epilepsy. Oppenheim doubts that disease of the sexual apparatus ever produced epilepsy, but if the aura starts from the site of an injury, the scar should be excised, if one can be found. Starr agrees with Oppenheim, while Dana says that it is now agreed that the removal of ovaries, although diseased, never cures a case of epilepsy.

It would seem that cranial injuries would prove a fertile field for curative operation. The sequence has been clear, and the injuries have been palpable; i. e., operation was suggested for epilepsy following a cranial traumatism, Jacksonian attacks, or grand mal attacks, in which there was a clinical evidence of a focal lesion. With all these definite indications it has been found that if the condition has existed more than a year or two, such a condition of nervous instability has been implanted in the brain that temporary arrest of the convulsions is about all that can be expected. Temporal decompression has been advised, but if good and permanent results do not follow one operation, it is not advisable to duplicate the procedure with the hope of a better result.

THERAPEUTICS. Homœopathic. In the indications for all remedies it must be understood that the basic indications for any remedy should be present in the case before the special indications are to be considered.

Agaricus. Where the attacks are characterized by the exertion of great strength. Dewey quotes from Winterburn the cure of a case with the 30th potency prescribed on the great flow of ideas, and loquacity after the attack.

Amyl nit. Has been recommended for use by olfaction, when the convulsions are preceded by marked flushing of the surface.

Argentum met. Seizures like epilepsy, followed by delirious rage, in which the patient jumps about, and tries to strike those about him. The probability is that the cases reported have been hysterical.

Argentum nit. It has helped some cases, and is particularly applicable to those occurring at the menstrual period, or from fright. The attack is preceded by dilatation of the pupils for days or hours before the attack. The attack is followed by restlessness instead of sleep, and there is general tremor.

Artemisia absinth. Is efficient in attacks of petit mal where consciousness is not entirely lost, where attacks are repeated many times a day, and where there is giddiness, with a tendency toward falling backward, and a persisting tremor. Seizures preceded by vertigo, an aura of a warm sensation rising from the stomach, and followed by a slight impairment of speech (Dewey).

Artemisia vulg. Another remedy which has been successful for epilepsy from fright or some mental emotion, where the attacks occur in rapid succession; also in typical petit mal, where the patient is unconscious only for a few seconds, and then resumes his occupation as if nothing had happened.

Arsenicum alb. This is advised in such cases as have, preceding the convulsions, a burning sensation in the stomach, pressure and heat in the back, extending to the nape of the neck and brain, with dizziness. The stupor following the convulsions is broken by restless agitation.

Belladonna. For acute epilepsy where the cerebral symptoms are prominent, where the face is flushed, and the whole symptomatology seems to picture cerebral irritation. There is an aura as if a mouse were running over the extremities, or of heat arising from the stomach. There are illusions of sight and hearing; the convulsions are apt to commence in an upper extremity, and extend to the mouth, face and eyes. The great irritability of the nervous system, the easily disturbed sleep, the startings, the tremors and twitchings, and general Belladonna symptoms, render the choice easy. Atropine has also been used successfully. (Dewey.) The author has used Belladonna with great benefit on the general indications, plus conspicuous visual aura, i. e., no convulsion without bright flashes suddenly appearing.

Borax. This remedy has been used without very precise indications, but is of value when a patient presents a general nervous impressionability. Nervous instability is its keynote.

Bufo rana. This is especially indicated where the attacks are in the young, are marked by great periodicity, and result from the debilitating effects of masturbation. The attacks are ushered in by a cry, and the face is livid. The attacks are preceded by a marked mental irritability, and the patient may be paraphasic. The aura is in the solar plexus. Spasms are better by warmth.

Calcarea carb. Epilepsy about puberty. Prescribed principally upon constitutional symptoms. "Peculiarly adapted to persons who withdraw themselves from all companionship, and tend to melancholia." (Dewey.)

Causticum. Menstrual epilepsy. Best suited to recent and light cases, and nocturnal attacks. Involuntary urination in the stage of stupor. Great faintness and weakness following the attack.

Cicuta vir. Convulsions are nocturnal, frequent, easily recurring from the slightest touch, and followed by prostration similar to that found in Causticum. Great faintness and weakness following the attack. The aura is replaced by a venous congestion, photophobia, and mental confusion, and the pulse is slow. The countenance is markedly cadaverous, and the jaws tend to become locked.

Cocculus. Convulsive attacks preceded by the characteristic symptom of hollowness or emptiness of the whole body, the head especially. Tinker quotes a prescription of Cocculus given by O'Connor for long-lasting petit mal. The indications were a feeling of clutching at the base of the brain on mental exertion, also eructations of gas even on attempting to read. Ten drops of Cocculus θ were put in four ounces of water, and doses were taken every two to three hours, until almost all had been taken; the bottle was refilled with water, and taken until the contents were reduced to about the 3rd dilution. Then a new tincture dilution was made, and the process was repeated for some months, with the apparent permanent cure of a very long lasting condition.

Cuprum met. An epigastric aura of long duration, with a most violent and continued form of attack. Highly useful in old and obstinate cases. The attack is preceded by signs of mental irritation, anger and irascibility; then follow the indications of a similar motor state; the fingers and toes twitch, and there is a chewing motion of the jaws. The attack itself is simply a developement of this motor irritability carried to the extent of general rigidity, and perhaps opisthotonos.

Cuprum acet. The 3x is reported to have cured epilepsy in a dog, with the guiding symptom of trying to scramble up a wall when the attack came on.

Digitalin. In doses of one grain of the 3x every other day, has been recommended for attacks arising from excessive nocturnal emissions.

Hydrocyanic acid. For attacks markedly tetanic in character. It has been termed by some a specific.

Hyoscyamus nig. Epileptic attacks preceded by the characteristic mental condition. There are twitching of the jaws, and a chewing motion, and during the attack a bluish countenance, and involuntary urination. Dewey says, "The convulsions have an hysterical character. They are the result of violent fright, and there are illusions of sight and hearing."

Ignatia. Violent convulsions with an alternately red and pale face. Suitable for young women, and for those who are of an excitable temperament.

Indigo. Where epilepsy is known to arise from worms, and the patient is of a sad, anxious disposition. Dewey says that Dr. Colby, of Boston, considers it superior to the Bromides; flashes of heat seem to arise from the solar plexus to the head, and there is an undulating sensation in the brain, similar to that of Cimicifuga.

Kali mur. This was Schuessler's standard remedy, especially for cases arising after the suppression of an eczema, or other eruption.

Lachesis. When the attack is preceded by cold feet, eructations, paleness of the face, vertigo with heaviness of the head, and palpitation of the heart. The patient screams out, and the limbs jerk, and the fists are clenched. Clinical results have been good in some cases.

Lycopodium. Epileptic attacks of the usual type, but with extreme periodical anxiety.

Melilotus alba. This was highly recommended by Dr. Bowen, but has not found very general endorsement. The indication is an extremely severe type of headache preceding the attack.

Nitric ac. Nocturnal attacks preceded by a sensation as though a mouse were moving up and down the left side.

Nux vomica. Such cases as seen to arise directly from indigestion. In the author's hands this has proved efficient in cases where the gastro-intestinal symptoms were conspicuous, together with a high degree of irritability of the nervous system generally, especially irritability of temper.

Œnanthe croc. This exhibits in its pathogenesis the closest picture of epilepsy, and has been highly praised by many experienced practitioners, and the writer himself adds his testimony to its efficiency in controlling attacks. It seems to act better in the 3x or 6x potencies than in the tincture, and cases of cure are becoming more and more numerous. General symptoms are sudden and complete loss of consciousness, swollen, livid face, frothing at the mouth, dilated or irregular pupils, convulsions with lockjaw, and cold extremities. (Dewey).

Opium. This has been recommended for nocturnal attacks, with violent convulsive movements of the limbs ,and symptoms of suffocation.

Plumbum met. The characteristic symptoms are a paralytic heaviness of the limbs before the attack, and, after a prolonged sleep period, a temporary motor paralysis of the lower limbs. A cure is reported of a case which always appeared at the menstrual period, with colic, obstinate constipation, and a sallowness of the complexion.

Secale cornutum. Is highly recommended in rapidly recurring paroxysms, with rapid sinking of the vital powers, and paralysis of voluntary motion.

Solanum Carol. A remedy which in some cases has wrought cures, and according to Dr. Paine, reduces the attacks. Halbert also praises it. Melancholia seems to be the indication, and also attacks at the menstrual period. (Dewey).

Physiological Therapeutics. Epileptic Status is a condition of the greatest gravity, and tends to endanger life itself. Joedicke treated nine patients after the following method, and all survived. The patient must be placed in bed in a quiet room, and given two or three tepid enemas. All exciting conditions must be avoided, as the idea is to reduce the sources of external irritation, and the internal intoxication. After the enemas 3 grammes of Amylene hydrate is given per rectum with 10 drops of Strophanthus to sustain the heart. If convulsions still continue, he repeats the dose in a half hour to one hour. Then he draws 50-150 c. c. of blood from a vein at the elbow, and infuses 100-150 c. c. of Ringer's salt solution under the pectoral muscles. Jousset gives under these conditions Opium and Belladonna in a low dilution, and claims marvelous results. Some salt of Bromine is the standard medication, and while Sodium and Strontium and Ammonium have been assumed to possess advantages over the Potassium on account of reduction of the acne, and less disturbance of the stomach, it is now considered to be rather a fanciful distinction. Spratling prefers Bromapin, which is pure Bromine in Sesame oil. This does not produce acne, and it may be used in an emulsion, and so be a good reconstructive. All authorities agree that large doses are a mistake, and that from 15 to 45 grains a day should be the maximum, and that the appearance of acne is a proof of Bromide intoxication, after which its efficiency is lost; therefore the dose should be just short of the point of the production of acne. Thomson thinks well of the addition of 1 grain of Antipyrin to every 2 grains of Ammonium Bromide. Inasmuch as the majority of the attacks are nocturnal, it is advised that the last dose at night be double the quantity of those earlier in the day. The drug should always be given in large quantities of liquid. Turner (of London) thinks that the Bromides are applicable to only about 50% of the cases. When they fail he makes use of a mixture of Potassium Bromide, Picrotoxine, and Antimonium arsenate. The Picrotoxine is applicable, as it is a vasodilator. He denies the value of Ceni's Serotherapy, even in the hands of its originator, but Gordon (of Philadelphia) reports four cases which have shown such improvement as to promise cure. In these cases 30 c. c. of cerebro-spinal fluid is withdrawn from the spinal canal of an epileptic, and from 3-5 c. c. is injected subcutaneously in the arm of a second patient. The cerebro-spinal fluid of no patient is curative to himself, but it is to another (Ceni says to which among others is not always certain). This subcutaneous injection is repeated two or three times a week until the curative effects would indicate a reduction of the frequency. The Bromides are continued until the need for their use is less obvious, and finally they are discontinued. One of these cases had no lumbar puncture, and therefore that was eliminated as a curative factor. In this relation, Tissot says that the increase of intracranial pressure setting in before a convulsion would theoretically indicate a lumbar puncture as a remedy, but as a fact the severity of the seizure is not minimized, although as large quantities as from 40-70 c. c. have been withdrawn. It seems that the increase of intracranial pressure is not the cause of the convulsion, and that lumbar puncture is not a remedy. The use of the glandular extracts has proven inefficient.

Borax was strongly advocated at one time, but is now out of favor, and its place is taken, as an experimental drug, by Calcium. Sabbatani found that soaking the cortex in a solution of calcium chloride reduced its excitability, and that a lack of the calcium salts in the blood was a large factor in the production of the attacks, if not the actual cause of the disease. Littlejohn reported in 1909 the successful treatment of a very severe case by administering 15 grammes Calcium lactate three times a day. Donath reports negative results with the drug even in heroic doses. It is still, however, one of the drugs in favor.

In January of 1909 Dr. Spangler, of Philadelphia, had his attention drawn to the report by Dr. L. E. Self, of Clamette, Texas, of a male, thirty-five years of age, epileptic for fifteen years, who after being bitten by a rattlesnake had been free from epileptic attacks since then, i. e., for a period of two years. Deducing that the venom might be utilized as a cure for epilepsy, he injected the dried glycerinized venom subcutaneously in doses of from 1-250th to 1-25th of a grain every five to seven days. In 1911 he reported 36 cases with generally favorable results. Other physicians had made favorable reports. It is a purely experimental treatment, and must be considered so at present. Dr. Wm. H. Thomson, taking the position that the exciting cause is always an irritant to the afferent (sensory) nerve, advises that all peripheral irritations should be first treated, that one of the most efficient of these areas of irritation is about the epiglottis, and the hurried eating of the epileptic brings it into action. He therefore advises applications of silver nitrate 10 grains to the ounce once a week to the larynx. He believes in stimulation of the peripheral nerves by the use of a pack each night made by saturating a sheet in hot water containing a teaspoonful of red pepper to the pint, and enveloping the body in this. The diet should be practically vegetarian, and the mastication be slow and thorough. He uses small doses of bromide at first, and believes that its efficiency is much increased by the addition of one grain of antipyrin to every two grains of bromide. Chloral is useful, but never in greater doses than 10 grains. Sooner or later he uses an intestinal antiseptic in all cases. He believes as strongly in the open air treatment in epilepsy as in tuberculosis. Dr. Thomson's theory of peripheral irritation has not met with general acceptation, but he claims exceedingly satisfactory results from his treatment, and I have personal knowledge of one case where under this treatment very frequent attacks were entirely suspended for one year.

HYSTERIA.

DEFINITION. This is a disorder of the mind, where there is a contraction of the consciousness, and a weakening of the powers of correct deduction from peripheral stimuli. The conscious mind seems to be weakened, and the subconscious dominates the functions of the body. Hence we find a disordered condition of nutrition, vasomotor and trophic irregularities, and disturbances of the special senses. Moebius has said of it, that it is a condition where ideas control the body, and produce morbid changes in its functions.

THE SYMPTOMS OF A TYPICAL CASE are some form of muscular paralysis or abolition of some general, or special sense-perception, with or without a perversion of the functions of mind or body, or of both. It is accompanied by disturbance of cutaneous sensibility on the trunk, as well as the limbs, and the areas are bounded in many instances by right lines, or are of whole limbs, or definite parts of them. The field of vision is diminished, and there is a reduction in the area, or a change in the order of the relative visibility of colors (dyschromotopsia).

DIFFERENTIAL DIAGNOSIS. This is from Psychasthenia, Dementia Precox, Multiple Sclerosis, Epilepsy, and Meningitis.

FREQUENCY. It is a common disease, and confined to no one race, but the Celt, the Slav, and the Israelite furnish the great proportion of the cases.

AGE. Most frequent about the age of puberty, and although it has been thought to have occurred as early as the second or third year of life, it is rare before ten, or after forty.

SEX. Formerly it was thought to be found in the female only, since the name indicated its supposed seat and cause, viz., uterine; but now it is known to occur in the male; also the greater part of the female cases are found among the wealthy and idle, while the male cases, often traumatic and major in type, occur more often among the poor and overworked.

ETIOLOGY. Heredity is to be traced in seventy-five per cent. of the cases, and is mostly from the female side. Where we find it specifically in the ancestors, the case developes early. The late cases, and in fact in most cases, the heredity is not direct, but indirect in the sense that any debilitating condition in an ancestor predisposes the descendant to hysteria. These ancestral influences may be any of the cachexias, or intoxications, or the practice of vicious habits, but a hysterical mother is prone to have a hysterical daughter. Such an influence will of course be intensified, if such taint is common to both parents, and therefore, consanguineous marriages are prolific of such offspring.

The causes are distinctly predisposing, and exciting. The most common predisposing cause is deficient mental discipline in the family. The person who was not as a child brought up to endure with composure the ordinary vicissitudes of daily life, will suffer inordinate nervous strain when brought face to face with shocks in adult life, and if in addition he has inherited a neuropathic taint, he will almost inevitably develope some neuropsychosis. Any unusual emotional strain will be sufficient to inaugurate the hysterical condition. The same cachexias and intoxications which have been spoken of in the case of an ancestor, will operate directly in some cases; masturbation has been judged to be an efficient cause, and secret alcoholism has produced it in adult women. Castration and the climaxis have been followed by it, and such also has been the result of traumatism.

Freud says that the cause of many, if not all hysterias, can be designated "psychic trauma." Every experience which produces the painful effect of fear, anxiety, shame, or of psychic pain may act as a psychic trauma. The hysteric suffers mostly from reminiscences.

Pathology. Hysteria is a mental disease, and must be so treated, but it must be remembered that its phenomena are produced by physical mechanisms. The investigator can find no anatomical changes, but that the symptoms are produced by functional disturbances of the cortical cells is probable. A nutritional fault is always present in these cases, and there is an increase in uric acid, xanthin compounds, and urobilin. These products are competent to provoke arterial spasm, and may explain its occurrence as well as a direct intoxication of the nerve-cells.

Symptomatology. Generally speaking, there are a disturbance of common sensation, disorders of the special senses, the developement of hysterogenetic zones, and, less often, convulsions of an epileptoid character. Some of these symptoms are more permanent than others, or more usually found at all stages, and so they have been placed in two distinct categories. The common, usual and earlier, are called Stigmata, and by other writers referred to as Continuous, while the later group, which are seen later and less frequently, but quite as permanent when once established, are termed Paroxysmal, or Accidents (Church). If either stigmata or accidents appear, they are apt to preserve the same form throughout the disease. The presence of stigmata has been so dwelt upon in descriptions of this disease, that it should be emphasized that all cases do not present stigmata, and that in their absence the condition must be diagnosed by exclusion, from the fact that the disease simulated by the hysterical person will markedly differ from the actual disorder, either in the combination of the symptoms presented, or in the order or rate of their appearance.

Stigmata may be definite changes in sensation, or power, or control of the operations of the viscera, motion, or of the processes of the mind. The change is generally a loss of tactile perception (anesthesia). It differs from that due to organic disease, in its distribution and outline. It is distributed in patches over the face, trunk and limbs, or of half of the body, or a single limb, or an extremity. In diseases of the peripheral nerves, we may find extremities anesthetic, but the trunk is spared, or until very late in the disease, while in diseases of the cord, the anesthesias will be as they are represented in the segments of the cord, and not scattered here and there, with no relation to such representation; while if from the brain, we shall have an antecedent condition to explain its presence. It will impress the observer, if the area is large, that the anesthesia is of such description as could readily be pictured in the mind, since we can readily imagine an anesthetic hand, or foot, a whole leg or arm, or a half of the body. The outline is occasionally peculiarly irregular, but in a vast proportion, the anesthesias are bounded by right lines, i. e., forming squares and rectangles; or if of an extremity, it will have an approximately straight border for its upper limit. This gives rise to the name of "Glove" or "Stocking" form of anesthesia. Such outlines are very diagnostic, when we recollect that the skin is innervated by the expansions of sensory nerves in such a manner that the terminals interlace, so that in organic disease normal sensation fades into anesthesia with a great indefiniteness of outline. These cutaneous losses are possible to all forms of stimulation, and the patient may fail to appreciate heat, cold, and pain as completely as he does tactile sensation. In some cases, we shall find one quality of loss in one part, and a different one in another. Another fact is that they may be localized, or shifted by suggestion, or magnets, true or false, or by any other measure that strongly appeals to the mind of the patient. The patient is unconscious of these anesthesias, and as they are possibly in minute patches, long search may be necessary to establish their existence. Again, it is to be noted that acts of precision of touch and muscular control may be done as perfectly with the anesthetic as with the normal member. Nor are these losses confined to the superficial structures. There are deep anesthesias affecting the joints and muscles, and the viscera. This gives rise to a sort of hypotonia, and also to a loss of the perception of fatigue. When this is present, the patient cannot state correctly the position of a limb if the eyes are covered, and cannot execute movements correctly, since they are in fact ataxic. The abolition of the perception of fatigue explains the phenomenon known as "Flexibilitas Cerea," a wax-like rigidity, which is seen in the cataleptic state, where the patient will hold a limb in an elevated position for hours, which would be an impossibility for one in health. The mucous membranes of the mouth, ear, eye, and rectum, and urethra are subject to the same losses. It is diagnostic, however, that it never attacks the irregular area of skin covering the coccyx and its neighborhood, the perineum, and the genitals. Anesthesia of this region is always organic in origin. There is an apparent loss of special sense if an organ lies in an anesthetic area.

While loss is the rule, there may be an increased response to peripheral stimulation (hyperesthesia). There may be pain where simply touch should be felt, or touching one area may give rise to pain in another, but it will be found that the pain upon light touch is lost, or not increased, when it is replaced by considerable pressure upon the part. We may also find cases which are the subjects of spontaneous pain. Such pains are apt to be localized in parts where injury has been experienced, or is apprehended.

In addition, among these perversions of sensation (paresthesias) are ill-defined numbnesses, coldness, burnings, and queer sensations. Globus hystericus is one, which is a sensation as of a ball traveling up the esophagus, when in typical form, and is by some attributed to spasmodic and systematized contracture of the esophageal muscles and by others said to be without known physiological basis.

Losses of the functions of the special senses are common stigmata, and sight is the one most likely to be perverted. Contraction of the visual fields is most often seen, but as it occurs also in neurasthenia, reversal of the color fields (dyschromatopsia) is the most diagnostic sign. A person in health ought to perceive an object brought forward along the side of the head, so soon as it reaches a position at right angles to the axis of the direct vision, i. e., he ought to have a visual arc of 180 degrees, less the loss on the nasal side. The hysterical patient suffers a diminution of this area, all the way to a field just the size of the aperture of the iris (tubular vision). This is often bilateral. To test this condition, seat the patient in front of the examiner, and have him fix his eyes directly on the eyes of the examiner at about twelve to fifteen inches distance. Then a vibrating pencil is to be held at arm's length at the side by the examiner, equidistant between the two persons, and, still vibrating, is to be brought slowly nearer and nearer to the patient, always keeping to the middle line between the two, the patient having been warned previously to say "now" as soon as he perceives it. By repetition on the other side of the head, a sufficiently clear determination of the field of vision may be obtained. By closing one eye, and moving the pencil forward upon that side of the head, the nasal area of vision, made narrower by the encroachment of the nose, can be mapped out. Do this upon both sides, to avoid over-looking half-vision (hemianopsia), a loss usually from an organic cause. Reversal of the color-field (dyschromatopsia) is discovered by the use of some form of perimeter, and rests upon the fact, that the normal eye perceives white over the whole visual area, blue over a little less extended field, red over still less, while green is the most contracted. In hysteria, this order is often reversed in an eccentric manner, so that, for instance, green may be visible over a larger area than blue. In such a case, however, the revolution of a Newton's Wheel, bearing red and green, will be seen as white, as in the normal eye, showing that the retina and eyeball are normal, and that the fault is in the brain. Where one-half of the body is anesthetic, sight will be lost, or will be very dim upon that side, complete loss being rare, but may come on suddenly, and go as suddenly, but the blind eye will be found to be used in binocular vision and prisms will produce diplopia.

Faults in accommodation may be a symptom, and we may get a diplopia, or covering one eye, the other may see double or triple objects. Visual losses being present, the conjunctiva, lids, and an area of skin about the eyes will be found to be anesthetic.

Hearing is liable to be perverted to a partial degree, total deafness, however, being almost certainly organic. As in sight, so here, the meatus and skin will be anesthetic. This deafness is not due to a defect in the auditory apparatus, but, as in visual losses, resides in the brain, and is shown to be so by Rinné's test. The normal ear will hear the vibrations of a tuning fork about twice as long when it is held in front of the ear, as when its heel is pressed against the mastoid process; that is, air conduction is better than bone conduction. A hysterical person gives the same response to the test, showing that the auditory loss is not in the ear, but in the brain. It is said that a hysterically deaf person, in attempts to hear, does not incline the head toward the speaker, as does one actually deaf.

The Voice may be lost, and in such case the larynx is insensitive. The organic and tendon reflexes, are not modified, as in similar anesthetic conditions from organic causes. The pupils will react to light and accommodation. The iris will dilate upon pinching the skin of the neck, even though that may be anesthetic, and though the sight may be diminished. The abdominal and cremasteric reflexes are normal, and if the skin is pinched where it gives rise to pain, the respiration and action of the heart will be accelerated (Mannkopf's sign.) Buzzard says, that the unusual combination of an exaggerated knee-jerk, and a diminished plantar reflex is common and diagnostic. An absolute loss of knee-jerk is never hysterical, but the ankle-clonus may closely simulate that arising from organic disease.

Palpebral and pharyngeal reflexes may disappear, if the parts are insensitive, but there is no constant rule, and they may be voluntarily restrained, and tickling the trunk or plantar surface does not produce the usual response. The pharyngeal is absent in 90 per cent. of hysterics.

Motor Stigmata. These should be carefully separated from the phenomena known as motor accidents, and they must be sought for, because the patient is ignorant of their existence, although practically they differ only in degree from the stigmata. Motor stigmata are very variable in their forms of expression, but commonly there is a tardiness in the commonest movements, which rate of response is greatly influenced by the diversion or concentration of attention upon them. They are also characterized by an incoördination in movement, which is, of course, proportionate to the loss of muscular sense. but is peculiarly independent of the presence of anesthesia, since, with an anesthetic hand, a patient can grasp a pencil even with the eyes bandaged, and indicate its use, although if the anesthesia is complete, there will be a loss in this power. Another feature is the inability to perform simultaneous movements, or those which require divided attention. Voluntary movements are also weaker than is normal, and if a limb has been strongly flexed or tightly bandaged or subjected to any treatment which strongly appeals to the mind, it may be the seat of rigidities and contractures, in which case it will be found that the tension is equal upon both flexors and extensors, contrary to the rule in organic complaints. Similar treatment may cure it.

Visceral symptoms may consist of spasms of intestinal or gastric pain, or dyspepsia or constipation, vomiting or regurgitation of food, and anorexia to such an extent that the patient becomes extremely emaciated, and may often be bedridden. Anuria is a very common stigma, and there appears to be a spasm of the renal arteries, for the patient may void little or no urine for several days at a time, but deception should always be suspected. It may be of value to remember that while retention and suppression of urine are common in hysteria, incontinence is not a symptom. Vasomotor symptoms are to some extent a universal phenomenon. There are flushings and pallor, fever and chill, extremities as cold as ice or burning hot. There is one type where the feet and legs are cold and white, and pit on pressure. There is also the blue edema of hysteria, reminding one of Raynaud's Disease, in which the leg is blue, and shows a reduction of several degrees in temperature, but does not pit, and, contrary to Raynaud's Disease, gangrene does not supervene. A rise of temperature is a common incident. There are two types: one where there is a continuous fever, and another, where there are temporary exacerbations of temperature. The temperature may differ upon the two sides of the body. Dissociation of pulse and temperature is common; with a pulse of 90, temperatures of 112 to 118 have been reported.

Mental Stigmata. These cases are often characterized by peculiar losses of memory (amnesia), and this loss may relate to certain portions of time, certain groups of facts, or the movements of particular people, and the loss may be so systematized as to lead them into very erroneous statements, and hypnotism will often show these to be purely functional losses. Weakened will power (aboulia) may exist, and it will give a person a hesitancy in performing the commonest acts, and an uncertainty in regard to the fact of their execution. All these people are extremely suggestible, which makes them ready subjects for hypnotism. They simulate every kind of known complaint and condition, not for the pleasure of it, but because they are the prey of an unregulated response to external stimulation, both material and psychic. Contrary to popular opinion, they are not erotic, as most hysterics are sexually cold.

Accidents. These, as has before been said, have some of the characteristics of stigmata, and, when thoroughly established, may have the force of stigmata, but, properly speaking, are sensory, motor, and convulsive attacks of a more temporary and paroxysmal character. Sensory accidents may take the form of a headache resembling that of syphilis or migraine, or that from tumor, or meningitis. There is a hysterical pseudo-meningitis, which resembles the organic disease in the intense pains, the wild cries, and even the opisthotonos, but is differentiated from meningitis by the absence of fever, and the presence of a normal cerebro-spinal fluid. We find all grades of irritability of the spine, and of neuralgia of the viscera, and there is a form of angina pectoris, which is considered to have hysteria as its base, and is not easily diagnosed from the organic disease.

Motor Accidents. The so-called paralyses are practically losses of energy to move a limb, and not loss or impairment of any of the mechanism to perform an action (abasia.) Contractures also exist, with or without the presence of pain. One type of this symptom is called Brodie's joints, where contracture is combined with a neuralgia, or hyperesthesia generally of the large joints. It is the most common of all the monosymptomatic attacks of hysteria. A diagnostic point is that the pain is not worse at night. Charcot says that it is diagnostic that after an anesthetic muscular rigidity returns first, and pain next. Charcot also says, that in malingering the production of a contracture requires great concentration of attention, strength, and energy, and that, from this exertion, we get great irregularity of movement, and respiration is increased, which symptoms are absent in the contracture of hysteria. The complete monoplegia of a whole member, or of only the hand or foot, with normal movements of the corresponding member, is found only in hysteria, and is either flaccid, or combined with contracture. In such case, the limb will show some contraction of the muscles if the corresponding limb is being used, or in intoxication, anger, or terror, and if the limb is placed in a position requiring voluntary action for its support, it will not immediately fall if support is withdrawn. Rhythmical spasms of limbs, or single muscles, are not uncommon.

Tremor is usually said to be passive, but instances are quoted where it is as much intention as that of multiple sclerosis. The etiology of the tremor found in hysteria is not always clear, since toxic poisoning, multiple sclerosis, and other diseases in which tremor is a prominent symptom, are not infrequently complicated by hysteria.

Hysterical Convulsive Attacks. Charcot's description is classical. There may be a prodromal stage of mental and physical symptoms, which are apt to be uniform for a given case. The symptoms may be depression, or increased irritability, fear, palpitation of the heart, globus, etc. Then comes what might be termed an aura, rising from the ovaries, from the stomach, or from the throat, combined with marked fear, tinnitus, palpitation, mist before the eyes, and mental confusion. Then we have: (1st) The epileptoid period, where the patient falls, not suddenly as in epilepsy, but still in a tonic condition. (2d) Period of clownism, which is inaugurated by clonic spasm, contortions, and grand movements, opisthotonos, etc. (3d) Passional period, of plastic attitudes, expressing devotion, or passion, or fear, with an expression of face conforming to the movements of the body. (4th) Period of delirium, of a quiet nature, with prominent hallucinations of animal forms. The whole duration is from fifteen to thirty minutes. but the attacks may succeed each other, so that the condition will last for days (the status hystericus). The exhaustion of true epilepsy may bring on an hysterical convulsion. It is to be diagnosed from epilepsy, from the fact that the tonic stage is longer, and opisthotonos is marked; the movements are more active, and betray volition, and consciousness is rarely completely lost. External influences increase the severity of the attacks; the corneal reflex is never lost, the light reflex is retained, and the eyelids may be seen to flutter. The tongue is very rarely bitten while the lips may be. While it is the usual rule that the feces and urine are not lost, Oberndorf (Med. Jour. Oct. 10th, 1914) says that immobile pupils, biting of the tongue, frothing at the mouth, and even incontinence of urine have been seen in hysteria. It is followed by a delirium like an anxious terror, which is as likely to follow a mild as a severe attack. There are modified and partial attacks, sometimes vertiginous, succeeded by lethargy.

Epileptoid Attacks, having only one or two phases of the grand attack just described, are more common phenomena, and in some cases are very difficult to differentiate from epilepsy. Attacks of tetany, of ecstasy, of sleep, or somnambulism, of noctambulism, or of catalepsy, may follow the epileptoid attacks. (For differential diagnosis see article on Epilepsy.) In catalepsy fixed positions are taken and maintained, as if the limbs were of wax. The heart and respiration are slow and weakened (Cheyne-Stokes respiration may be present), temperature is normal, or slightly subnormal; sensibility and reflexes are lost; we may have involuntary movements from the bladder and rectum, but the corneal reflex is almost always present. These attacks may last from hours to months.

Aphonia, which remits, comes on from emotional excitement, passes away spontaneously, or from some agent similar to that which has caused it, is a very common symptom. There is a form called aphonia spastica, which may be so sudden and severe as to demand tracheotomy. In this condition, the vocal cords are only partially movable, but it is diagnostic that the impairment is never unilateral. There is a form of dyspnea, which occurs in hysteria, which comes from a sudden laryngeal spasm, or spasm of the diaphragm, or there may be a simple increase in the rate of respiration—from 60 up to 100.

Mutism. This is rare. It is diagnostic, that while the person understands everything, he is more mute than aphasic. There may be all sorts of sounds in the larynx; the person may talk backwards, and have all sorts of perversions of speech.

In addition to these somewhat systematized phenomena, there is a variety of lesser manifestations of the hysterical state. Trophic and vasomotor accidents are shown in the production of bullæ, and other eruptions, in sudamina, and bloody sweat. Painful swelling, and those characterized by an increase in tissue only, without sensory symptoms, have been noted by many observers, of which hysterical breast is a type of one variety, and phantom tumor of the other.

DIAGNOSIS. On the positive side, we may consider a case to be hysterical, if we find, in combination with symptoms of apparently organic origin, the various stigmata which have been described in the preceding pages; or if the disability in question has not the history, or distribution, or order of evolution which we have a right to expect in such conditions. On the negative side, optic neuritis, or atrophy, loss of knee-jerk or Achilles-jerk are always of organic origin. So are decided nystagmus, hemianopsia, Babinski's reflex, sustained or prominent ankle-clonus, motor or sensory paralysis in a single nerve or nerve root, incontinence of urine, except a temporary loss in spasm, and muscular atrophy with the reaction of degeneration. The mental state of hysteria is never diagnostic. A new pupillary test has been presented to differentiate between organic and psychic pain. If the pupil is contracted by the presence of a strong illumination, pressure upon an organically painful area will result in a relaxation of the pupillary ring, but will not result if the pain is of psychic origin.

Differential Diagnosis. "Multiple Sclerosis cannot certainly be differentiated until the typical symptoms of nystagmus, scanning speech, and intention tremor have appeared." (Starr.)

Meningitis. Headache, vomiting, convulsions, and rigidity occur at times in hysteria, and are usual in meningitis, but there are also symptoms of a toxic condition, which are conspicuously absent in hysteria. In a case of tuberculous or serous meningitis the diagnosis is difficult, since cold douches may, for the time, have as salutary an effect upon the one as upon the other condition. The variations of temperature in tuberculous meningitis are so slight that they may invite error, but the stigmata of hysteria are absent, and the pupils are stiff, or irregularly dilated, and some grade of optic neuritis is present, changes not found in hysteria. The spinal fluid is clear in tuberculous meningitis, but in it are lymphocytes and flocculi, and it coagulates in a peculiar thread-like coagulum, while no such change can be found in a case of hysteria.

Neurasthenia. Is distinguished by the inability of the patient to endure sustained exertion of any kind, mental or physical. This exhaustion reveals itself in the exaggeration of the knee-jerks, and other deep and cutaneous reflexes (lack of inhibition), while in hysteria the response is paradoxical, i. e., one is perhaps exaggerated, while the other is reduced. An exaggeration of the knee-jerk and a reduction of the plantar is quite common.

Dementia Precox. The catatonic stage is differentiated from hysteria by the fact that in the latter there is to be detected slyness and method in the contrariness, and purpose in the actions. They are tyrannical to those caring for them. In the catatonic there is an evident senselessness and lack of purpose in the movements, and the emotional activity exhibited. Finally the hallucinations and delusions are more exaggerated and prominent in the catatonic.

Psychasthenia. Hysteria may present inhibitory symptoms which would remind one of psychasthenia, but they are not fixed to such a degree, nor are there the phobias, indecisions, and irresistible impulses found in psychasthenia. Moreover, the psychasthenic recognizes the nature and foolishness of his limitations, while the hysteric does not, because the compelling delusions are beyond his conscious control. Paget said of hysteria: "It says 'I cannot not,' it looks like 'I will not,' but is really 'I cannot will.'"

PROGNOSIS. The prognosis for recovery is always good, but the return to health may be very slow. Recovery from the paroxysm, of whatever nature it may be, does not always mean recovery from the disease, and the recognition of this fact is the essential for the happy issue of the case.

TREATMENT. In the consideration of treatment it must be remembered that hysteria is a mental disease, and that the treatment here outlined is only a framework upon which a successful therapeusis is to be built up. Some cases are very simple in their psychical dissociation, and are speedily restored to health by any shock, operation, or specific remedy. These are the cases which in old times were treated by having a pail of cold water thrown over them, which has been replaced in these days by cold douches in the bathroom with equal success. These are the cases cured by a fortunate prescription, by circumcision, by operation on eyes, nose, or other assumed source of reflex irritation. Lumbar puncture is a good therapeutic procedure, as it will clear up the possibility of a meningitis, and if repetition is promised if the symptoms persist, it will often abort a cataleptic or resistive phase. Convulsions, paralyses, rigidities, and neuralgias of great severity have been cleared up never to return by the hypodermic administration of 1-20th to 1-10th grain of apomorphia, for the almost immediate attack of vomiting is a powerful deterrent to future exhibitions, since there is a tremendous difference between posing as an object of universal sympathy, and being simply ridiculous. At times, if the bed is screened in, so that the patient can be under observation without her knowledge, and no attention is paid to her bizarre demonstrations, she will promptly recover. In cases of aphonia and mutism, Bartlett has advised a bungling anesthesia which will cause the patient to struggle and cry out, and so disclose the inorganic nature of the symptom. The same procedure is to be advised in other conditions.

Malnutrition is usually very evident and the repair of the body will often be followed by the return of the mind to the normal. If the disease occurs in a robust, well-nourished patient, change of scene may be sufficient to work a cure. The treatment, however, in the two types of cases, does not differ so widely as might at first appear, for change of scene does not necessarily mean travel, nor can malnutrition be combated only in a sanatorium. The patient is always courting attention, and has a morbid craving for sympathy, and a removal is often a *sine qua non* for recovery. She, for in ninety-nine cases out of a hundred the patient is a woman, has been brought up to be idle and self-indulgent, and we ask a great deal if we expect the indulgent parents to stand by and assist us in what seems to them to be harsh and unreasonable treatment. Therefore removal from the family, if only to another street in the same town, is often necessary. The Rest Cure of Weir Mitchell probably depended for much of its success upon the personality of the physician, but it is very beneficial to selected cases. The tendency in isolation is toward introspection, which is an evil, and in some cases an unconquerable one, but generally it will be more than balanced by the absence of opportunity for spectacular display. I have seen seclusion alone work a prompt and lasting cure. If we decide that the patient is a proper subject for the rest cure, she must have massage, to get the benefit of passive exercise, and to equalize the peripheral circulation. She should be fed liberally, and frequently; by a nasal tube, if she will not take food otherwise.

General faradism is a powerful adjuvant, both for effect upon the circulation, and for its suggestive value, and it should be given strong enough, so that the patient will be glad when the treatment is over. Cold sponge baths, alternate hot and cold sponging of the spine, are all valuable, and should be modified according to the class of symptoms presented. If the patient sleeps poorly, a bath of 100° for ten minutes at bedtime, or the hot pack, may be of assistance. The combination and manner of application of these elements in the treatment are as much a matter of good judgment as the selection of the elements themselves. Our aim should be so to fill up the day, that the coming of night will be welcomed, and yet neither mind nor body should be exhausted in the process. A plan like this is often feasible:

7:30 A. M. Patient, while still in bed, should have a cup of cocoa.

8:00 A. M. Rise, and take a cold sponge over the whole body; standing in warm water to the ankles, if the shock is too great.

8:30 A. M. Breakfast in bed, and if the case is one of malnutrition, the nurse feeds the patient.

9:00 A. M. The patient sits in a chair, preferably in another room, while the bedroom is being cleaned for the day, and during this time may be read to from paper or book, or indulged in conversation.

10:00 A. M. Massage and electricity for one hour, then a cup of hot milk or broth.

11:00 A. M. to 12:00 M. Room dark, and patient in bed, and alone, but under supervision.

1:00 P. M. Dinner.

1:30 to 4:00 P. M. Conversation, a little reading, the patient may walk about the room a little, after the first week. Cup of hot milk, or broth.

4:00 P. M. Nap until 5:30, when she is dressed, talks and amuses herself until supper-time.

6:30 P. M. Supper.

7:00 P. M. Conversation until 8:00.

8:00 P. M. Gentle massage, followed by an alcohol rub, warm bath or hot pack according to circumstances.

9:00 P. M. In bed for the night.

During this period, no letters are to be written or received; no visitors, and only the most brief calls, at long intervals, from members of the family.

One great object is occupation for every moment, and after the first physical and mental protest, it promotes physical quietude and mental equanimity. As the patient grows better, the nurse should take up with her a regular system of calisthenics, and when still better, she should begin to walk in the open air, irrespective of weather or season. Just as a pair of scales is of great assistance in stimulating the patient to increase her physique, so a pedometer will stimulate her to walk increasing distances, until she will easily cover from eight to ten miles a day. If exercise has a bad effect upon the symptoms, it must be reduced temporarily, and if the walking runs up the pulse or the temperature, it must be discontinued for a day or two, and then recommenced. The patient generally becomes interested in her physical developement, and *pari passu* drops her mental vagaries. To the extent of her powers we should add weaving, basketry, brass work, modeling, and out of door sports. Healthy physical tire promotes healthy sleep, and a proper action of the bowels, which latter must be the subject of most careful attention, since intestinal toxemia perpetuates and increases the trouble. In regard to this function, a little time spent at stool, with a good deal of massage and electricity are more beneficial than cathartics. The ingestion of a large amount of water is uniformly beneficial. Tympanites may be so excessive as to impede the action of the heart, and here faradism is often beneficial; or the abdomen may balloon up into what is known as "phantom tumor." Under anesthesia this will at once disappear.

ELECTRICAL TREATMENT. King doubts the purely suggestive effect, and traces many failures to reliance upon this, leading to the ignoring of the proper technique. The static bombardment may do good, it is true, but, while hysterical aphonia may be instantly relieved by the sharp pain of electrical shock, it is oftener cured by passing a current through the larynx from side to side. The best treatment is to obtain the catalytic, and nutritive effects of the current upon the brain and spinal cord, as well as upon the diseased part, or member. Pass a galvanic current longitudinally and transversely through the head, then through the cord, in the same way. For motor losses, the faradic current is the better, but for sensory changes, the static spray is usually preferable, yet galvanism may act when both the other currents fail. A hyperesthesia should be treated with a soothing current, and anesthesia with a strong static spark, or faradic brush. Neuralgia of this kind is usually to be treated with the galvanic current, but may be better relieved by the faradic. Two diagnostic points will thus be elicited. If a strong faradic current is not felt over a patch of anesthesia, the cause is an organic one, and not hysterical at all. While either pole of the galvanic battery is equally potent to relieve a hysterical neuralgia, it is not true, if it has an organic basis, since in that case the anode relieves, the cathode aggravates. For clonic spasms, place the positive pole on the spasmodic muscle, current gradually raised, and as gradually decreased. This applies also to paralysis of the viscera. In hystero-epilepsy, the convulsion may be stopped by energetic electrical treatment, the variety of current not being important. The chief value of electricity in hysteria, however, is to build up the patient between attacks, rather than to treat attacks, and this is to be done by galvanism through the head and along the spine, general faradization, or the static breeze. Treatment should be given daily. Hysterical contractures have been relieved by the static spark, and elevation of temperature, salivation, and polyuria, may be relieved by the same general treatment. As a rule the treatment must be symptomatic, aiming at the removal of hemianesthesia, paralysis or contracture, localized neuralgic pain, asthma, globus hystericus, etc. Use such a method as will give the electrical result which you desire, and also strongly impress the patient, and, therefore, the faradic brush, and static spray or spark are preferable to galvanism.

Every one of these measures is good, and the skilful combination of them is the only road to success, and yet they only furnish the conditions under which the personality of the physician can readjust the twisted mentality. In some way we must bring to the surface the mental injury, injured feelings, wounded pride, shattered affections, or consciousness of sin which is the cause of the disease, and show its trivial nature, or allow it a normal outlet, and then the patient will recover. This may be done by hypnosis, or by suggestion in the waking state, or in sleep, or in the helpful suggestion by a stronger character, but in some way it must always be the source of cure in all but the trivial cases. As this subject is far beyond the scope of a book of this type, the reader is referred, among others on the subject, to "Modern Treatment of Nervous and Mental Diseases," by White and Jelliffe, for a most reasonable and discriminating resumé of these modern conceptions of treatment.

THERAPEUTICS. Although no remedy can be expected to cure this disease, it may yet be the essential third factor to make efficient the hygienic and suggestive measures which are a necessary antecedent. Remedies are believed in by the patient, and demanded by the family, and we weaken our position by denying their efficiency, and taking away hope. We are forced to give drugs in the convulsive attacks, but the most favorable time for therapeutic action is between these attacks, or in their absence. If we can control the convulsions by our drugs, we go a great deal further than relieving an uncomfortable condition, but owing to the conditions surrounding these cases, selection and administration are often unsatisfactory. In such a protean disease, almost any drug may be demanded at some phase, but the following are most useful.

Ignatia is the great hysterical remedy, as it presents all the fickleness of the disease in its symptomatology; the patient is sad, depressed, indifferent, and yet is wonderfully impressionable. She laughs and laughs, finally ending in a scream, and then weeps to the same extent. She does not crave sympathy like the Pulsatilla patient, but is given to brooding and introspection, yet is as intolerant of contradiction as is the person helped by Chamomilla. The motor system is as much disordered as is the mental, and there is incoördination of movement, and in the functions of the body. The sensory apparatus is in a condition of irritable weakness, there is great physical sensitiveness, and a propensity to spasms and twitchings of muscles. Clavus is common, which symptom is also found in the Thuja and Coffea patients. Hysterical paralyses may be helped by this remedy, since they are the result of auto-suggestion, and not of organic changes. The sympathetic system betrays its implication by alternate flushings and chills, by globus hystericus, which symptom is also prominent under Asafœtida, and by flatulence, and other symptoms of gastro-intestinal disturbance.

Tarantula Hispan. In Ignatia, we were speaking of a remedy which acts upon the whole nervous system, while Tarantula affects the mind far more exclusively. The patient is a malingerer, and is full of cunning. All the limbs are in constant motion, and there is a great wildness of movement. Jousset considered the remedy to be of as much value as Ignatia, especially if convulsions were present. Headache is continuous, and there are frequent fits of laughter, which is like Theridion, but the latter is exceedingly sensitive to noise. Mygale lasidora is also like, but is especially for those cases which have chorea-like twitchings. The patient is very nervous, feigns symptoms, and it therefore suggests itself as a remedy for hystero-epilepsy. The patient appeals for sympathy constantly, while the Ignatia patient lives in a world of her own, and only resents intrusion. The Tarantula patient indulges in unusual forms of spasm if she cannot attract attention in other ways. The physical restlessness is said to be relieved by music.

Moschus is useful in cases where vasomotor symptoms are greatly in evidence. There are fainting, syncope, and globus hystericus, and the symptoms are often carried to the point of apparent suffocation. There is a simulation of tetanic spasm, which is to a less degree found in the pathogenesis of Asafœtida, Cocculus, Ignatia, and Nux moschata. In a recent case, the patient a girl of seventeen, held her breath until suffocation seemed so imminent that artificial respiration was repeatedly instituted. There are loud eructations, the patient is very nervous, has uncontrollable laughter, and scolds, as does the Palladium patient. Castoreum is somewhat like. There may be nymphomania or satyriasis. The drug acts best in the lower potencies, and is efficient by olfaction.

Kreosote has its principal sphere in those cases which are marked by very obstinate vomiting.

Platinum is imperious, with loud laughter, and boisterousness which borders on insanity. There is nymphomania, without the desire to uncover seen in Hyoscyamus. Palladium resembles this, but in the latter drug there is the fear of being slighted.

Valeriana. There is a state of general nervous excitement, and the patient must keep continually moving, and there are pains simulating rheumatism. Nervous agitation, with a sensation of warmth rising from the stomach is the key-note. The patient is joyous and lively, and has flushes of heat.

Nux moschata is adapted to nervous hysterical people who rapidly change from grave to gay, but its great feature is sleepiness with intestinal bloating, and a dry mouth. The patient is irritable, faints easily, and is exhausted by the slightest exertion. Such a case is a borderline between hysteria and neurasthenia. It has been strongly advised for the treatment of globus; Bayes advises for this a drop of Rubini's Camphor on sugar every 5-10 minutes (Bartlett).

Pulsatilla. The condition is one of depression and weeping, and the patient characteristically implores the sympathy of others, instead of indulging in silent grief. Changeableness of symptoms is another characteristic. It is commonly called for in the hysteria of puberty with menstrual derangements, the flow being delayed and scanty. There is constriction of the throat to the extent that speech is sometimes impeded.

Sumbul is advised by Barnes for acute hysteria, and given in doses of 30 minims of the fluid extract (Bartlett).

Silicea has the same sensitiveness to the opinion of others that was noted in the symptomatology of Ignatia, but is more actively whimsical and fault-finding. There are great irritability and motor restlessness, and it is particularly indicated for cases which have arisen from a shock, or exhaustion of the nervous system.

Chamomilla should be selected on its well-known mental symptoms, and is especially indicated by the persistence of joint-pains (Brodie's Joint, which is a pulpy degeneration of the synovial membrane and ligaments, mostly seen in the knee), which are sometimes a characteristic of the case.

Convulsions as a symptom may be controlled by any of the foregoing remedies, but may need more specific treatment.

Belladonna is indicated when a strong and vigorous patient developes wild spasms and convulsions, with a red face, staring eyes, pulsating carotids, and other evidences of vascular excitement.

Stramonium is for those more nearly simulating an epilepsy, with foaming at the mouth, and a fierce and maniacal condition of mind, giving rise to absurd fancies, loquacity, and so forth.

Hyoscyamus is more hallucinatory than the previous remedy, and the convulsions may be of a milder type. It has nymphomania, with a desire to uncover the person, but this probably arises from cutaneous irritation.

Gelsemium has few marked hysterical symptoms, but has convulsions, with spasm of the glottis. There is mental irritability, with semi-stupor, languor, and vascular excitement. Sensation of a lump in the throat, which cannot be swallowed. It is a good remedy for male and female onanists. Vesical irritability with a constant desire to urinate. This remedy will sometimes meet almost all the indications in a case where vascular depression, instead of exaltation, is a prominent characteristic.

Actæa racemosa. This has the temperament of Ignatia, but with more or less infra-mammary pain. Uterine symptoms are prominent, with irregular and delayed menses, and there are neuralgias. Hysterical attacks with delirium, the patient jumps about, and there is loquacity with constant change of subject.

These remedies may all fail in the convulsive attacks, from the difficulty of selection, or administration. Between attacks an attempt should be made to meet the particular deviations from health which are presented by the individual patient. In addition to the remedies referred to above there is frequent use for Lillium tig., Sepia, Lobelia inflata, Asafœtida, and Sulphur.

CHAPTER XV.

SPASMODIC DISEASES.

THE MYOCLONIAS.

This chapter includes the diseases which are distinguished by the presence of twitching spasms of all kinds:

Chorea of Sydenham, or Infectious Myoclonus.
Huntington's Chorea.
Tics.
Paramyoclonus Multiplex.
Electric Chorea of Bergeron, Hénoch and Paget.
Fibrillary Chorea of Morvan.
Myoclonus of Unverricht.
Electrical Chorea of Dubini.

CHOREA.

ST. VITUS DANCE, CHOREA ANGLORUM, CHOREA VULGARIS, SYDENHAM'S CHOREA, INFECTIOUS MYOCLONUS.

DEFINITION. Since the time of Sydenham a group of pathological nervous symptoms has been known by this name. It has been described as a spasmodic affection of the nervous system limited in duration, and characterized by a jerky incoördination of voluntary movements, involuntary twitchings accompanied by some degree of physical and mental weakness, varying largely in different cases. In true chorea the movements are involuntary, purposeless, and liable to intensification under the influence of emotional excitement, and cease during sleep. It is at present a fairly debatable question whether it is not a symptom arising in the course of various infections rather than a disease.

Electric Chorea, Huntington's Chorea, Tics, and Habit Spasms have a very different etiology and symptomatology, and will be described under separate headings.

THE SYMPTOMS OF A TYPICAL CASE. A person, most probably female, in childhood or early youth becomes physically weak, mentally irritable, and developes irregular jerking movements of members which should habitually combine in the execution of purposeful and emotional movements. The jerky incoördination may involve all parts of the body, and in many cases, in severe ones always, involves the muscles of the tongue, face, and lips, and interferes with phonation and deglutition.

DIFFERENTIAL DIAGNOSIS. This disease is to be differentiated from Metallic Intoxications, Toxic Poisons, Hysteria, Multiple Sclerosis, Friedreich's Ataxia, Athetosis, Post-hemiplegic Chorea, Huntington's Chorea, Tics, Myoclonus.

RACE. Is almost exclusively Caucasian, and cases have been said to be especially frequent in the upper classes, but the somewhat wide experience of the author indicates that privation and faulty hygiene cause as large a number of cases among the poor.

SEX. Two or three females to one male.

AGE. Rare before four years, or after twenty. An enormous preponderance before the fifteenth year.

ETIOLOGY. As a preliminary to a discussion of the etiology it must be remembered that the normal child is a creature of constant movement; of movement for the sake of movement, on account of the stimulation of the nervous centers from the vital turgescence of growth. Inhibition is not so potent as in later life, and therefore every child needs only a slight increase in nervous irritability to exhibit choreic movements. Such a factor may be added by any one of a long list of intoxicants and infections, whereupon the child is then called "choreic." If neurotic in heredity, the chance is by so much the greater, and so we find evidences of nervous instability or dissipation with great frequency in the ancestors of these cases. Direct heredity is more rare, but the disease was traced for five generations in one family, appearing in all cases under the age of thirty. One child was immune when he had arrived at the age of fifty, and it was interesting to learn that he alone, of all the family, was not nursed by the mother. That the predisposing cause is a nervous instability, and not an organic defect, is pretty conclusively proved by the almost universal recovery of even the most severe cases, and, therefore, lesions of the central nervous system found post mortem must be considered as results, and not as causes. Poor hygienic conditions, mental strain, reflex irritations, infections, intoxications, and perverted blood-states may all act as exciting causes. Rheumatism is a common infection, but not so exclusively as was formerly believed, but whether rheumatic or other infection; Ponthière says the avenue of entrance can so commonly be proved to be the tonsils, and diseased conditions of the pharyngeal vault, that disease of structures in this locality is classed as an exciting cause, and he has invariably cured his cases by removal of these diseased glands.

PATHOLOGY. While no distinctive pathology can be stated, and its existence is seriously doubted, it must be kept in mind that there is not an entire agreement with this statement. Richardière and Lemaire found a lymphocystosis constantly in the cerebro-spinal fluid of five boys and ten girls, between the ages of five and fourteen, who were suffering from typical chorea. One case seeming negative was found to be not chorea, but a special neurosis in the young child of a mother who had suffered from eclampsia. Other observers have reported the same meningo-encephalitis, but still others have with equal certainty disclosed an absolutely antagonistic pathological picture, so that we are forced to deny that any constant pathological picture has been presented.

SYMPTOMATOLOGY. There is usually a prodromal period of malaise, insomnia, restlessness, peevishness, poor appetite, constipation, and mental dulness. The latter is the most common mental attitude, but the emotions may be too readily excitable. The patient's general condition is poor, and the hemoglobin may be 60 or lower. Next is to be seen an inability to preserve a steady contraction of the muscles. It may be only a quivering of the eyelids, or a lack of dexterity of the hands, but usually in all cases there is an unsteadiness upon the feet. Movements may be voluntarily suppressed for a time in mild cases. Most marked of all, but often unobserved, is the readiness with which fatigue is induced. We may find bone or joint pains, and the premonitory malaise continues. The muscular movements resemble voluntary motions, but they are purposeless, and are jerky, and begin rulably in the hands, thence go to the forearm, and perhaps affect the face. The disease may begin on one side, and go over to the other. The method of the protrusion of the tongue is diagnostic, in that it is snake-like, and the lips being also affected, speech may be very much affected. The decrease of muscular power is very marked. In some cases the chorea, while very severe, is confined to one half of the body (hemichorea). The sphincters are never involved, except in the last stages of fatal cases. Electrical excitability is often increased, and may show a moderate degree of reaction of degeneration. In the grave types we are apt to find very marked pains in the joints, and evidences of endocarditis, with elevation of temperature, and rapid pulse. Some of these cases have been brought to autopsy, and have proved to have been septic endocarditis from streptococcus. In these cases the patient throws herself from side to side continuously, and with great violence, and is unable to make herself understood by speech, is unable to feed herself, and at times is unable even to swallow food if presented to her.

In these cases rectal feeding is the only possible procedure. Mental fatigue is easily produced, and we get capricious emotional expression, and also stupidity; there are hallucinations and delirium of some grade, and this is quite the rule in adult cases, and those complicating pregnancy. There is a form of chorea in which mania supervenes, and overshadows the motor symptoms, and is called Maniacal Chorea. Sachs says that it is a simultaneous development of a mental and choreic disturbance in the course of a toxic or infectious process. Such a case is reported by Finney, in which the maniacal outbreak was preceded by rheumatic pains in the legs for a fortnight, with a mitral systolic murmur. Chorea of pregnancy, Chorea gravidum, in twelve out of fifteen cases is found to have been preceded by a like affection occurring at the time of puberty. It usually occurs in a primipara, appears generally about the third month, and may be so slight as to consist in only a few spasmodic movements, or become so violent as to lead to a fatal issue from inanition, and lack of rest. The symptoms usually disappear in three or four months, but may recur at parturition or lactation. The danger to the mother is not great, but may imperil the life of the child, as abortion, and premature labor are not unknown results. These patients are apt to suffer from hallucinations. Sometimes cases go on to paranoia. Two cases of Dementia choreica have been reported. These cases had the walk of paralysis agitans, and the spasms were of a major type, with minor intermixed. There was no rhythm, and they acted like jumping-jacks. One of these cases was traumatic. The mental symptoms were irritability, seclusiveness, history of persecutory ideas, characteristic speech defects and progressively increasing dementia.

COURSE. An attack usually lasts about four weeks, and one third of the cases show recurrence, to which females are more liable. These recurrences are usually three in number, each one being shorter, and the interval between being longer than the preceding one. The last remaining symptom is a twitching of the face, or protrusion of the tongue.

PROGNOSIS. Recovery is the rule in cases occurring before the tenth year, but there is a fatality in plain uncomplicated cases of 2½%. When it thus occurs, autopsy sometimes fails to reveal an adequate cause. Complicated cases die from exhaustion from excessive muscular effort, inability to take food, endocarditis, and other heart complications, cerebral hemorrhage, concurrent affections. The chorea of pregnancy is fatal in 25% of the cases.

VARIATIONS ON THE ORDINARY TYPE. Chorea Gravis, or muscular insanity, which is more fatal, since the violence of the spasms exhausts the patient, and prevents the taking of nourishment.

Choreic Status where, like Status Epilepticus, the spasm continues for days, and the patient may die of exhaustion.

Limp Chorea where paralysis is the most marked symptom; monoparesis is the common form, the muscles are lax, reflexes are abolished, but atrophy is rare.

DIAGNOSIS. Depends upon the developement of irregular, involuntary, and incoördinate movements of all varieties, even while at rest, beginning in the face or an extremity, and extending over a considerable portion of the voluntary muscles; diagnostically affecting the tongue with a jerking, darting motion on attempts at protrusion, and not being preceded, or accompanied by evidences of an organic brain or nervous disease. Lymphocytes are frequently found in the cerebro-spinal fluid.

DIFFERENTIAL DIAGNOSIS. Intoxications from poisonous metals or toxins may appear like it, but the muscular fault in such cases is a tremor, rather than a jactitation, and there are associated symptoms from the primary disease.

Hysteria gives rise to rhythmical, not jerking motions, and there are usually stigmata, or a history of antecedent seizures; the patient is apt to be older, and there is a history of a psychical causation.

Multiple sclerosis has similar movements, but they are volitional. While the eyes twitch in chorea, there is no defined nystagmus.

Friedreich's Ataxia. This has an unsteadiness on the legs and in gait, which simulates the gait and station of chorea, but the peculiarity of incoördination does not appear while the patient is seated, and at rest. The deep reflexes are apt to be diminished, and the disease is familial.

Athetosis. The movements are slow and even, and not jerky, although they do appear while the patient is at rest. There is a history of some antecedent causative disease.

Post-hemiplegic Chorea. This has the history, and the diagnostic marks, of a preceding hemiplegia or diplegia. There are spasticity, and increase in the deep reflexes.

Huntington's Chorea. Always familial, and seldom begins before middle life, slowly progressive, and terminates in dementia.

Adult and Senile Choreas are not allied to chorea causitively, but are the results of senility, or pre-senility on a neuropathic heredity.

Tics or Habit-Spasms. These are of single muscle-groups, and show no tendency to extension.

Myoclonus is usually a disease of adults. It is also usually located, or begins, in the lower extremities, and is bilateral, the face being spared as a rule. The muscular movements are very rapid, reaching the rate of 60 to 100 per minute, and a tonic stage may supervene.

TREATMENT. Rest in bed is the best of all measures, as exhaustion is such a pronounced factor; this is peculiarly true of cases from the lower walks of life, where the disease is often due to improper hygiene, and lack of proper food. Isolation is also necessary in severe cases, and forced feeding, massage, and Swedish movements are valuable in the disease, and especially valuable in neurotic children, for the purpose of prevention. Oppenheim says that sixteen hours sleep out of the twenty-four should be procured. A good method to follow in severe cases is this: in the morning give an early breakfast of crackers and milk. One hour later give a bath at 100°, or a pack at the same temperature. The temperature of the pack is to be gradually lowered by adding cold water to the pack; then pack and cover are all wrapped in a warm blanket for fifteen minutes. The blanket is then removed, and then the pack, and the child is rubbed down with cold water until the skin is in a glow. An hour after this a more substantial breakfast is given, and a little quiet play is allowed. After movements have ceased, the rest and careful hygienic treatment should be continued for an additional two weeks. Medicines are of subordinate importance. On the other hand accurate prescribing may obviate the necessity of any especial hygienic care, other than the avoidance of exhaustion, and is especially called for in cases where hygienic conditions have been good. It is not so certain as the first method, and severe cases will demand the skillful exercise of all the means at our command. In those severe cases called Muscular Insanity, where the patients throw themselves about continually, and with great violence, it is necessary to pad all the projecting parts of the bed; and all the joints of the patient. Even this will not always obviate abrasions and bruises, and in such cases I have sometimes found that the best results will be obtained by placing the patient upon a couple of mattresses spread upon the floor, where the amplitude of the space seems to have a good effect upon the violence of the movements, and also takes away some possible sources of injury.

Electrical Treatment. The role of electricity is as a general nervous tonic. There are no pathological changes with definite localization for treatment. Almost all children are terrified by the apparatus, and the sensations produced. General faradic brushing might be of some advantage if such treatment appealed to the patient, and the element of fright could be eliminated. King's advice is to the point: a mild static treatment with negative insulation only, allowing the charge to pass off by the hair and other points, the charge being a light one, and sparking being carefully avoided, with later a breeze along the spine. Certain habitual choreic movements, which remain after the general twitching has passed away, may be removed in a week or two by a static spray over the spine. He also states that a form of choreic movement which occurs in adults, and does not respond to general treatment, is not benefitted by any form of electricity. This is naturally true, since it is not a true chorea, but the result of organic changes arising from cerebral palsy in childhood.

At the Children's Convalescent Home at Chappaqua, out of a registration of 250 they are liable to have one-third choreics; some of them slight; some very bad, and some who have shown improvement, and then halted. They instituted there, some time ago, a regimen which included plenty of sleep, plenty of plain food, sports in the fresh air, and systemic massage of the muscles, and have had a suprising increase of cures, the massage being the deciding factor.

THERAPEUTICS. Agaricus muscarius. This remedy is one of the most frequently indicated in neurotic children, and adults who have indulged excessively in tea-drinking. There is a mental element in the symptomatology, as is shown by the idiotic look. There are emaciation, and sensitiveness of the spine, especially in the lumbar region, and blueness of the face and the extremities, with such paresthesias that the patient complains of itching and stinging in the skin like frost-bite, and they suffer from chilblains. Trembling, jerking, and restlessness may continue during sleep. The facial muscles, eyelids, and eyeballs are implicated in the movements, and there may be angular contortions of one upper extremity, and of the opposite limb in the lower part of the body. By preference I have used Agaricin 2x, 1 grain powder every two hours.

Arsenic. It is to be thought of in stubborn cases, where uneasiness is a marked symptom. There is a constant desire to change the position of the legs, and to walk about for relief. It corresponds well to the chorea of debilitated children. It has mostly been used in the form of Fowler's solution.

Actæa racemosa. This is a remedy which should be thought of where the symptoms are notably rheumatic or myalgic. There are sharp lancinating pains in various parts of the body, there is marked soreness of the muscles, and the spine is tender to touch, and is the seat of burning sensations. There is a tremulousness of the whole body, nervous restlessness, insomnia, and mental depression. The action of the heart is irregular, there is a catching pain, and all the symptoms, both physical and mental are aggravated by excitement, and relieved by rest and warmth. Good for cases occurring after eruptions (like Natrum mur.), for rheumatic girls, and for women with pelvic disorders, or those where there is menstrual irregularity, or aggravation during menstruation. The movements are more apt to be on the left side, and the fugitive pains, endocarditis, and insomnia are (according to Dewey) the characteristic symptoms.

Belladonna. This remedy is distinguished by the intensity of the muscular incoördination, the excitable disposition, and the marked vasomotor symptoms. Dewey states that it is to be thought of in the grand choreas found in dentition and pregnancy. In the latter case the disorder is plainly toxemic, and a most vigorous eliminative treatment must be inaugurated without delay, as these cases often prove fatal. The drug has been used both in potency and in the tincture.

Calcarea carbonica. In cases where malnutrition is very evident, and fright or masturbation has been an exciting cause it may be a proper selection. In these cases speech is difficult, as the tongue is bitten when talking. O'Connor quotes a very severe case cured with the 200th.

Causticum. The indications for its use are that the patient is rheumatic, that the effects are mostly shown on the right side of the body, and that, since its effects are quite strikingly localized in the muscles of the face, tongue, and lips, although the arms and legs are not immune, it is necessary for the selection of this remedy that speech should be affected. The motions are said to continue during sleep. While Butler and Dietz report very striking results, Jahr insisted that it was only useful in minor cases which showed no mental impairment.

Cicuta virosa. This was advised by Allen in case spasms occurred during the course of an attack of chorea. It has seemed to me that its action is so peculiarly spinal, and on the medulla, that it is not a remedy for chorea itself, but might be called for in view of some intercurrent condition.

Crocus sativa. Exceedingly neurotic patients with hysterical twitching of single muscles, and more of a hysterical than a choreic symptom-complex.

Cuprum. Baehr said that this drug would cure most cases. It is indicated when the spasm starts in the muscles of the fingers and toes and extends to the limbs. The patient is better while asleep, but when he awakens the terrible contortions and awkward movements recur. Saltatory spasms may occur (Dewey). Both the metallic and arseniated form of the drug have been used by me with success.

Gelsemium sempervirens. It is called for in severe cases of chorea major when we find the constitutional symptoms. Some alternate with Aconite with good effect. Dewey quotes Gregory, of Yerka, California. He claims that he cured eighteen cases in succession with this drug. He considers that it is not efficient except in doses of 5 drops of the tincture of the green plant every 4 hours. This may be efficient, yet it is not Homœopathic, but sedative and antispasmodic, and Trional is a better drug for such a purpose.

Hyoscyamus. Both mind and body are affected in these cases. There is muscular twitching and trembling; the arms and legs, and both sides of the body are equally affected to such an extent that gait is tottering, and the patient can use neither his hands nor legs efficiently. With this is associated a hallucinatory state, which has the peculiarity that impressions of distance are distorted, and the patient reaches for distant objects as if they were near, and the contrary.

Ignatia. It is given on general grounds, much as is Strychnia in the Old School. It is actually a hysterical remedy, psychical instead of physical, and since we know that chorea is undoubtedly physical, the field for Ignatia is entirely in those hysterical cases which are so often confused with chorea.

Laurocerasus. Emotional choreas after fright; the contortions are fearful while the child is awake, and sleep is restless. The child can neither sit, stand, nor lie quietly on account of the jerking. Speech is indistinct (Dewey).

Magnesia phosphorica. This is indicated in the cases characterized by a tendency to neuralgia, as well as spasmodic affections.

Mygale lasidora. Recommended by many authors for severe cases with great motor excitement, but has always failed in my hands (Butler). A perfect picture of chorea. Twitching and jerking of facial muscles; one arm in constant motion. Hemi-chorea (Dietz). Useful in the light and semi-hysterical type of case, and here it is very dramatic in its effects at times.

Nux vomica. Advised for chorea with spinal symptoms; dragging feet, unsteady gait, crawling sensations in the ailing parts, and constipation. If the paresis persists Cocculus is advised. Nux vomica is much used, but it probably is valuable only as a general nerve tonic like Strychnia.

Pulsatilla. When the peculiar mental condition exists, and there is associated with the disease menstrual suppression or irregularity, and when it recurs in young girls at the age of puberty (Butler). Functional cardiac disorders (Cobb). Often indicated (Dewey). Simultaneous invasion of the joints.

Tarentula Hispaniensis. This is for the severest cases. There is a general muscular incoördination, often preventing the patient from walking, dressing, or feeding himself. The worst cases I ever saw yielded rapidly to this drug in a perfectly marvelous manner (Butler). Hysterical cases. Right arm and leg affected. The causes are fright and grief. There are restless movements at night. The patient is restless, must move constantly, the spine is sensitive, and there is trembling. Can run better than walk, and is useful in chorea major where there is rigidity. Cases are relieved by mental diversion, particularly by music.

Veratrum viride. Chorea major with severe muscular contortions, marked arterial excitement, and irregular action of the heart (Butler). Violent congestion to the nerve centers, and an excited pulse (Dewey). In plethoric girls.

Zizia. Useful if cerebral circulation seems greatly at fault. Movements during sleep.

Under some conditions it is advisable to begin the treatment with some constitutional remedy, and Calcarea carb. has been mentioned as one of these. Sulphur and Psorinum may be thought of in this connection; Phosphorus for children of a tuberculous diathesis, who have grown too fast; and China for children who have broken down nervously from masturbation, and other losses of fluids. Other remedies to be studied are Argentum nit., Asafœtida, Asarum, Cina, Cocculus, Dulcamara, Ferrum, Iodine, Phosphoric acid., Rhus tox., Santonine, Sepia, Spigelia, Zincum met. and Zincum phos.

Physiological therapeutists formerly confined themselves to Arsenic, Ergot and Asparin, to the practical exclusion of all others. This list has now been enlarged so that at the present time many prefer Trional, and use some Antipyrin with success. It has been pointed out that Arsenic is not free from dangerous effects upon the nerves and kidneys, and urged that the urine be examined frequently, and that neuritis and mental disorders be kept in mind. Recently Salvarsan has been used as a form of arsenical therapeusis in some cases of all grades of severity, with prompt effects. Weill and others have recommended arsenious acid as superior to arsenic in solution, and it is given in powders of thirty times its bulk of sodium chloride, and each powder is, for administration, rubbed up in 10 grammes of butter, and spread upon bread. From the first day the patient gets an increasing dose up to the sixth day, and then it is reduced proportionately daily. Arsenic is almost always given in the form of Fowler's Solution, and is begun with an initial dose of from 3 to 5 drops, 3 times a day. It is increased a drop a day, until toleration is exceeded. This is shown by slight nausea, or pain in the stomach, or puffing under the eyes. It is never best to push it to the extent that vomiting occurs. When the symptoms of tolerance occur, one or the other of two courses is pursued. One method is to wait until these symptoms subside, and then begin again with the original dose, while the other is to begin with the dose which was being administered when the toxic symptoms first manifested themselves. Of these, the first plan is the better.

Riviere treats all cases, except those in which control only can be exerted, as follows: He gives for one dose, Fl. Ext. Ergot, 1 drachm—Liquor Arsenicalis, 3 drops; increase this as may be needed. Trional is given in doses of 5 grains, 3 times a day, for a child over four years of age.

Asparin is well borne by children, and a child of thirteen years can take 10 grains every 4 hours. The drug should always be given in a cold, acid medium, and never in an alkali. While it may be given in milk, it is best administered in a dry powder, or in cold lemonade.

Tull states that in a most severe case, fifteen years of age, all the usual remedies, including arsenic, had failed to relieve the continuous and violent motion. He then gave hypodermically Apomorphia gr. 1/40, and within three minutes the child slept quietly and peacefully; gr. 1/20th by mouth was then given every 3 hours with arsenic, and within ten days the child was well, and without any sign of nausea.

ADULT CHOREA, CHRONIC PROGRESSIVE CHOREA.

HUNTINGTON'S CHOREA.

DEFINITION. It is a rare disease, first described by Dr. Waters, of Franklin, N. Y., in 1841. Dr. Gorman made it the subject of his graduation thesis in 1848. Dr. Lyon published the history of three cases in 1863, and Dr. Huntington described it in 1872. His name rather than that of his predecessors has been attached to it because he pointed out the three diagnostic symptoms of late invasion, heredity, and mental deterioration. It bears no relation to Sydenham's Chorea, except that it manifests incoördination of movement. The movements are slow, and very incoördinate; it attacks persons between the ages of thirty and forty-five, is familial, is slowly progressive, and does not tend to a fatal termination, but to mental deterioration.

AGE. Huntington thought that it was exclusively after the age of thirty, but Freund reports the case of two brothers who were attacked respectively at the ages of six and twelve years, and Lang reports one at twenty-six.

SEX. Males are slightly more liable to this disease than females.

ETIOLOGY. It is hereditary, and runs through several generations, but if it skips a generation, it never reappears (Huntington). Its transmission is through both the male and the female lines. Boyd reports six cases out of seven children of a choreic mother, with a choreic brother. Succeeding cases tend to develope at an earlier age.

PATHOLOGY. A case studied from Greppin in 1892 furnished the following findings: there was a pachymeningitis, and leptomeningitis; the roof of the skull was sclerotic. The brain appeared normal macroscopically. The microscope showed in the gray and white matter of the brain, throughout the whole organ, well-marked accumulations of cellular elements, always aranged in foci, which had proliferated into the healthy tissue, entirely filling the innumerable pericellular and perivascular spaces, and causing atrophy of the ganglion cells and nerve-fibres. Whether parenchymatous degeneration, or vascular alteration is a primary condition is doubtful. Lorenz examined the spinal fluid in two cases, with negative results, both chemically and microscopically.

SYMPTOMATOLOGY. The movements usually are slight, and of few muscles, beginning in the hands and arms with restlessness, or sudden movement of the part, which looks like mere nervousness. but slowly increases, involving the face, lower limbs, and body, until finally speech becomes indistinct and broken, locomotion uncertain, and attended by falls, mastication and deglutition difficult, and the exact and coördinate use of the hands is impossible. In the face and hands the movement consists of a sudden and more or less violent contraction of a muscle or group of muscles, momentary in time. and followed by complete relaxation. This is almost at once succeeded by an equally strong contraction in a distant muscle or group, or the two may be simultaneous; as the face and one hand, or one hand and the hips. The movements attack the flexors almost exclusively, are painless, extend over a far wider range than chorea, and the slowness of oscillation. and constant increase in the number of muscles involved is characteristic. The movements cease during sleep. In the hand the fingers alone may be affected, or the whole hand may be drawn to the ulnar side. The erratic movements may be under the control of the will in the early stages, and the patient may be able to thread a needle and to write. The movement in the arm of one patient jerked the hand from the lap nearly to the face. The gait may become dancing or staggering, and there is a wild incoördination. The patient carries the feet far apart, and after taking a few steps in an awkward manner, he may raise one foot abnormally high, and then bring it down firmly on the ground. Then stooping suddenly, and bending the body slightly forward, with hips pressed back, he raises himself on his toes, and while rocking in this position to regain the natural one, he goes through many contortions of feature and gesticulation. Regaining his balance, he then throws his body forward, and after taking a few steps comes to a sudden halt. In this way he gets about to some extent, although with frequent falls. The movements of the face affect principally the inferior and intermaxillary spaces, the upper face and eyeballs rarely being affected. There is no marked emaciation, nor evidence of circulatory or metabolic change. The mind is clear, although the features give a look of mental dullness, as the corners of the mouth are drawn downward, and outward. Mental change is reserved to the terminal stage, when unsoundness developes in the form of a progressive dementia. In literature it has been rather the rule to find that the mental change first appeared as an overexcitability or melancholia with suicidal tendencies, sometimes with delusions of grandeur, lasting for two or three months, to be followed by mental weakness with minor delusions. It is peculiar that the suicidal tendencies, as a rule, have been abortive, and that there has been a suicidal strain among the patient's ancestors. Sleep is sound, but the pain sense is dulled. There are no sensory, nor electrical changes, and the reflexes are normal. Duration is from twenty to twenty-five years. A few cases have been rapid.

Diagnosis. This depends upon the *first* appearance of the peculiar symptoms late in life, a gradual developement of slow incoördination of movement, with a history of ancestral disease of a similar type. The mental changes come at too late a period to be useful for diagnosis.

Differential Diagnosis. To be diagnosed from Adult Chronic Chorea. This has a cause in pregnancy, hysteria, or some disease of the heart or blood-vessels, and does not have the slow onset, nor the heredity. Others are Athetosis (which has a wavy contracture, beginning and ending slowly, and followed by rigidity), Sydenham's Cho[illegible], Ataxia of Paresis, Disseminated or combined Sclerosis, Senile Chorea.

Prognosis is good as to life, but hopeless as to cure.

Treatment. This is mainly hygienic, but it has lately been suggested by Hope that from its similarity to general paresis it might be benefitted by anti-syphilitic treatment.

TIC.

Definition. Brissaud defines Tic as a "physiological act, functional and purposeful in character, which has become a habit, purposeless, and meaningless in its execution." The movement may be simple, of an eyelid, for instance, or it may be of several muscles, or become generalized over the muscles of the body. The muscles involved will be found in some cases to be symmetrical, as in blinking, indicating a cortical source of the impulse, and if of more than one group of muscles, will be those physiologically associated for the performance of function, which indicates that the initial impulse was volitional. The generalized tics are almost always epidemic, and are exemplified in the Maine "Jumpers," the "Latah" of Malaysia, and the "Myriat chit" of Siberia ("play the fool," as these persons will execute all sorts of movements at command). It is always the resultant of a mental and muscular defect in the individual. To sum it up: it is a spasmodic neurosis, cortical in origin.

Age. Tic usually begins in childhood, or its antecedents can then be discovered, but it may come on at any age, up to maturity.

Sex. Not a factor.

ETIOLOGY. Initially the subject for tic suffers from an acquired neurasthenia, or psychasthenia, or comes of a neurotic ancestry, the stock which is prone to exhibit a fundamental weakness of will, a lack of inhibitory power. They are usually psychasthenics who give credence, subconsciously, to morbid impulses and obsessions. They are also defective, often less obviously, in the stability of the motor mechanism, and therefore if such a person is exposed to a mental shock, he may react in motor expressions of defence or replacement. It is much more common, however, for a tic to grow up as a very obvious effort to remove or neutralize a physical irritant, or to replace or cover up this primary reflex by a second one which will not attract attention to the actual act of neutralization. An irritation of the conjunctiva causes one to wink, or the irritation of a tight collar induces one to twist the neck. Soon it becomes a common resort from any irritation, mental or physical, and a habit is formed, which the person, weak in inhibition, finds it difficult or impossible to correct. It is always basicly volitional, and since it is so often symmetrical, it must be cortical in origin. The initial irritant may be purely men[illegible], but it is usually physical, and may be weak eyes, adenoids, or even an ill-fitting article of dress. The lack in the training of a child to ignore or resist unpleasant conditions, permits trivial annoyances to inaugurate a tic. Dana states that these movements sometimes represent an abortive attack of chorea, or are the residuum of an old attack. They are at times mimicry.

PATHOLOGY. Tic is a psychoneurosis, and the pathology is inferential. Since any psychoneurosis with a basis of heredity implies a weakness upon the mental side, and also in the apparatus of its expression, we may conclude that the presence of a tic depends upon an inherited defect of developement in the cortex of the brain so that the cells are unstable, and the association tracts poorly developed. These persons are not insane, although obsessed, but insane persons often develope tics.

SYMPTOMATOLOGY. The movements of tic are abrupt, and in the mild cases are a simple single movement of one set of muscles, but generally they come on in attacks which are apt to last many minutes, in some cases hours, and at other times the muscles are so convulsed for days that the patient becomes exhausted. The periods of recurrence may be very irregular and variable in degree, the convulsive stimulus being confined to a small branch or even a single twig of a nerve. While some movements are simple, and so remain, the tendency is to introduce complication and involvement of other groups. The movements may be tonic or clonic. Combined with the obviously muscular spasm, there may be unrelated acts, as of sniffing, or a clicking in the larynx. Later these sounds are produced by the simultaneous spasm of the glottis and the diaphragm. Stammering is not a tic, but is often an associated symptom in cases of tic. In addition, a fairly common adjunct is the ejaculation of a meaningless word, or an oath, or an obscene word (coprolalia), or the repetition of the remark of another (echolalia), or the imitation of movements (echokinesis). It is a diagnostic characteristic of these movements that they may be restrained by the will, but that such restraint is unpleasant, or even painful to the person, since there grows up within the person a certain kind of pleasure in the tics. The movements usually cease during sleep. The reflexes may be exaggerated, but generally reflexes, sensation, and sphincters are unchanged. The affected muscles may hypertrophy. While all muscular groups may be affected, there is a relative rule in their distribution. Tics of the face, head, and neck are the most common, and of all these, blinking the eyelids of both eyes is oftenest seen. Tics are usually in a combined form. The muscles of the eyeball may be affected, producing nystagmoid tic. All sorts of combinations may be found in the tics of the facial muscles, and the tongue is also affected. Grimaces are tics of the lips. A very common tic is a rotation of the head by a spasm of the sterno-cleido-mastoid muscles, generally associated with a spasm of the trapezius. The head may be drawn to one side so that the face lies on the shoulder. Both neck muscles (the sterno-cleidos) are more rarely affected, and the head may be drawn forward. There is sometimes a tic of the posterior muscles of the neck (splenius capitis) which draws the head backward. These tics of muscles of the neck are apt to cause considerable pain in the muscles. Spasms of the arms flex them, and draw them across the thorax. Tic of the hands and fingers is rarely seen, and spasmodic contractions of the extremities still less often, and when they do occur they cause jumps, kicks, and change of steps. Spasm of the abdominal muscles gives rise to the so-called salaam spasm, or nodding spasm. Maladie de Tic Convulsive, Gilles de la Tourette Disease, is the name given to a generalized form of tic developed in definitely neurotic individuals, which follows a quite definite course of evolution. After a severe mental shock a tic of the eyelids, or a facial musculature grimace, slowly establishes itself. It next extends to the neck and shoulders, and finally involves the trunk and extremities. Speech and respiration are affected, giving rise to grunts and groans, and all the varieties of involuntary ejaculation referred to previously. Obsessions and abnormal impulses grow up, delusions are developed, the mind progressively weakens, and finally dementia developes. The patient is conscious of the impending attack, and tries to avert it by a series of movements, like buttoning his coat, or counting his steps, but it may burst through this defence, or only so much escape as an ejaculation (Dana).

Brissaud speaks of what he calls mental torticollis, which the patient can check by merely resting the finger on the chin. All through the symptomatology of tic the lack of normal inhibitory power is seen. The person is ashamed of the bizarre movements, the coprolalia, and dreads the pain of the twisted neck, but he fears them less than the pain of the struggle to restrain them, and there is a sort of satisfaction derived from the movements, so that in some cases, after a period of restraint for some reason temporarily imperative, the tiquer is apt to indulge in a perfect debauch of unrestrained spasm. The inhibition of these attacks is variable in degree, and is hardly ever complete. Cold, fatigue, exhaustion, tend to increase the convulsive activity, while perfect comfort aborts it. Mental stress never fails to excite it.

PROGNOSIS. The prognosis of a case of tic depends a great deal upon the degree to which the patient will co-operate with the physician. Most cases recover under proper treatment, the Tic Convulsive, and that of the aged having the worst prognosis. Some cases continue throughout life, and remissions and change in type are not indication of a cure. If a child is defective, the prognosis is bad, and likewise if he is untrained and undisciplined. If, on the contrary, his character is being properly developed, the outlook will be favorable, and growth and developement in themselves have a favorable influence, Oppenheim reporting a case of recovery in a young girl at her first menstruation. Recurrence is probable.

DIAGNOSIS. The diagnosis of tic rests upon the discovery in a neuropathic person of muscular contractions, abrupt and irresistible in character, systematized in distribution, not in conformity to the distribution of a definite nerve, but combining muscles physiologically related for the execution of some movement or set of movements, with a definite period of rest between them. The movements, initially, are always for a definite purpose, and are only pathological because they are exaggerated in extent, and are now purposeless, since the cause of their original appearance has long since passed away.

DIFFERENTIAL DIAGNOSIS. It is possible to mistake tic for Jacksonian Epilepsy, but the very frequency of its repetition should speedily demonstrate the difference.

It might lead one to think of Chorea, but chorea is in unrelated muscles, there are no long periods of rest except in sleep, and generally the contractions of chorea are more widespread and violent, and cannot be imagined to be purposeful. The muscular contractions are not repeated in the same order.

Muscular Spasm is most often a source of doubt, but spasm is not purposeful, the contracted muscles following the distribution of some nerve; spasm is not at all under the restraint of the will, it does not cease during sleep, and is not confined to those of a neurotic heredity.

TREATMENT. Surgery is not a remedy. It is at times made use of in consequence of confusing tic with spasm. Surgery leaves the tic patient locally weakened by the operation, but consitutionally just as certainly a subject for some other localization of the neurosis. Psychoanalysis is the only remedy for the peculiarly psychic cases, and is helpful in all forms. Hypnosis may be an aid, but these patients are hard to hypnotize. The best results are obtained by a good mental and physical hygiene, followed by periods before the mirror, in which restraint of the movements is attempted. Electricity is generally considered to have only a psychic effect, although Hartenburg advises faradic treatment of the antagonists. Children should be urged to indulge in games which require accuracy of movement, and I have made use of checkers and backgammon, and also fox and geese, in this way. Placing pegs in indicated holes in a pierced board is one method of building up muscular control. While hygiene and education in restraint are the most efficacious agents, remedies sometimes seem of benefit.

THERAPEUTICS. Graeme Hammond has for a long time advised in spasmodic torticollis, Fl. Ext. Conium in gradually increasing doses up to the developement of its physiological action. The initial dose is five drops three times a day.

Cuprum arsenicosum is the best remedy, according to Bartlett, to which he adds Hyoscyamus and Agaricus, and if contractions are of a gyratory character he advises Strammonium; Jousset advises Lycopodium, Tarantula Hisp., Zincum, Sepia, Argentum nitricum, Hyoscyamus, and Laurocerasus.

SALAAM TIC. SPASM NUTANS. NODDING SPASM.

DEFINITION. This is a peculiar spasm which has been included among the tics by some, and by others among the epilepsies.

AGE. It is a disorder of early infancy, occurring almost always between the fourth and tenth month of life, or more nearly, between the age of first dentition and puberty. The children are unusually bright and precocious.

SEX. This is not a factor.

ETIOLOGY. It has been ascribed to rickets, and it certainly is more liable to attack children housed in quarters where there is a lack of light, and fresh air. It is not a result of inherited syphilis, or of birth injuries.

PATHOLOGY. This is unknown.

SYMPTOMATOLOGY. The spasm may be a repeated slight turning of the head upon the neck, most often a repeated nodding of the head upon the chest. This type may be replaced by more generalized movements of the whole body, or may share in the contraction which at first caused a nodding of the head only. In the cases occurring between the first dentition and puberty there is a convulsive salaam movement of the whole body, caused by extremely strong contractions of the abdominal muscles. This type, and according to some authors, both this and the foregoing types, are at times preceded by pallor of the face, staring and dilatation of the pupils, and there may be a loss of consciousness. Some authorities say that this is an epilepsy (Starr among others), others that it developes into an epilepsy, while still others (Coleman and Collier, for instance) say that epilepsy never developes, that it has no connection with the disease, and that the salaam form is merely a severe form of head-nodding. Mental deterioration may come on later, and result in idiocy, but some cases recover.

PARAMYOCLONUS MULTIPLEX OF FRIEDREICH, ELECTRIC CHOREA OF BERGERON-HÉNOCH AND PAGET—FIBRILLARY CHOREA OF MORVAN—MYOCLONUS OF UNVERRICHT.

DEFINITION. All these diseases or types of myoclonus vary in some degree from one another, yet are based upon a common characteristic. The patient suddenly or insidiously becomes the subject of short, rapid, unsystematized contractions of the muscles, preferably of the lower extremities, sometimes of the upper, but the face is spared. These contractions are clonic, and come in rapid succession (60-100 per minute), proportionately more as the muscle is smaller. They are irregular, arythmical, and do not involve the whole, but only part of a muscle, and are apt to involve symmetrical muscles. These contractions are not under the control of the will, but they cease during sleep. Irritation of the skin to the slighest degree will set up contractions, while vigorous use of the muscles rests them and quiets them. The muscular contractions are the only evidence of physical disturbance, since all the functions are normal, as are the reflexes, and electrical excitability.

ETIOLOGY. All forms of myoclonus are allied to hysteria, and are based upon a nervous heredity. They have been known to come on from overwork, after fright or infectious disease.

PATHOLOGY. Paramyoclonus multiplex of Friedreich is based upon a hyper-irritability of the cells of the anterior horns of the spinal cord. Similar spasms have occurred after removal of the thyroid gland.

Electric Chorea of Bergeron-Hénoch is quite common in children, and is characterized by the rapid, clonic, rhythmical contraction of any of the muscles of the body, so intense as to prevent the patient from following any occupation. The attempt to restrain them is futile, except to aggravate them. More than any other form this lies under the suspicion of hysteria, and is often cured by suggestion. As it is almost certainly accompanied by symptoms of indigestion, it has been considered to be an auto-intoxication, and improvement in the assimilation of food has been followed by a cure.

Fibrillary Chorea of Morvan, or Myokimia. This is peculiar in presenting fibrillary contractions, usually in the extremities, but no part of the body is immune. The symptoms are increased by irritation, and there is a decrease in the electrical irritability of the muscles.

THE MYOTONIAS.

Myotonia, or Tonic Spasm, is the designation of diseases which present tonic spasms which are symptomatic of organic disease.

Tetany.
Myotonia Congenita, or Thomsen's Disease.
Myotonia Atrophica.

TETANY. TETANILLA.

Definition. This is a disease characterized by painful cramps of a bilateral type, involving symmetrical muscles, beginning in the extremities, especially in the hands. These contractions are intermittent or persistent, and are associated with a mechanical and electrical overexcitability of the nerves and muscles. The disease may be subacute or chronic, epidemic, endemic, or toxic, and usually is based upon a condition of malnutrition.

Age. It is usually a disease of childhood, and is extremely rare after the age of fifty. In males it is most often found between the ages of fifteen and twenty-five, while in females the critical time is during pregnancy and lactation.

Sex. Among children, females predominate, while the reverse is true in adult life.

Etiology. It is apparently the result of a toxin, but perhaps not always the same one. It is said abroad that shoemakers and tailors are peculiarly prone to it, and it may be that there is an occupation toxin in those trades. It is endemic in Paris, Berlin, Prague, Vienna, and Syria (Church). Epidemics have been observed. It has followed typhoid, grippe, scarlet fever, measles, malaria, rheumatism, and many other infections, the puerperium, and lactation. Intoxication by alcohol, chloroform, morphine, ergot, and lead have produced it. It can be experimentally and clinically proved that it follows extirpation of more than five-sixths of the thyroid gland, and always follows extirpation of the parathyroids. Rickets, osteomalacia, intestinal worms, and gastro-intestinal diseases generally are the infantile causes, and especially where there is a gastric ulcer, with the resulting pyloric stenosis with gastric dilatation, and usually hyperchloridia. Langmead calls attention to a tetany due to dilatation of the colon. The local irritation of the passage of the stomach tube, or passage of a sound, or percussion of the stomach, cold, emotion, or overexertion are all competent causes. Tetany has been seen in children in connection with albuminuria. Most cases occur in the earlier months of the year.

PATHOLOGY. The results of autopsies are contradictory in most particulars, but two conditions are fairly established. There is a condition of congestion in the anterior horns of the spinal cord, similar to that found in poliomyelitis, and the parathyroids have been found to be the site of a great variety of degenerative changes. Peters claims that in seven cases he found an extradural inflammation of the connective tissues and fat, which constituted a pachymeningitis externa, and a root neuritis and ganglionitis.

SYMPTOMATOLOGY. A typical attack begins gradually, with a painful contraction of both hands simultaneously, and the result is a flexure of the hands in one of the following diagnostic types: the writer's hand, with fingers applied to one another, as if the patient were holding a pen; or the accoucheur's hand, with the flexed fingers tip to tip, and the thumb applied to the tips of the fingers; or when the interossei and the lumbricales are the site of the spasm, we find Main en Griffe, or "claw-hand" The seizure is usually preceded by numbness and tingling in the extremities to be affected, and at times by generalized symptoms of malaise, nausea, and less often by mental depression, vertigo, and headache. If the contraction spreads to the arms, they are flexed, the forearms are adducted and applied to the thorax, while the involvement of the legs presents them stretched out, that is overextended, and the feet also, while the toes are flexed, and the foot is inverted in an equino-varus position. The contraction may not be in the extremities alone, but in some cases the muscles of the trunk are involved, and while opisthotonos is sometimes produced, emprosthotonos (bending forward) is more common. The muscles of the neck may be so involved as to force the chin upon the sternum, or the dorsal muscles may as strongly retract the head. The muscles of the face may contract in facial spasm (trismus being a late symptom, if ever present), and the ocular muscles are not always spared. The muscles of the thorax are sometimes affected, and the diaphragm and the larynx generally are affected at the same time, and then the patient is threatened with suffocation; but serious attacks of dyspnea are generally in infantile cases, from spasm of the laryngeal muscles. The muscles of the tongue are sometimes implicated. The sphincter of the bladder in some cases has been thrown into such strong contraction that there is retention of urine, and the bowels are obstinately constipated. All sorts of variation in the number of muscles involved, and the severity or frequency of the attack may be seen. The spasm may be so slight that voluntary efforts can overcome them, but as a rule voluntary efforts to move the affected part are futile, and increase the pain. The cramps are tonic, not clonic, and they may last for minutes, hours, or even days. They do not cease at night, but may awaken the patient from sleep. The attacks may come on every few moments, especially in the epidemic form, or at intervals of some hours, or days may intervene. The attacks are painful, and there is generally a loss of sensation in the parts affected at the time of the attack. Fibrillary tremors are present in the muscles during contraction. Fever is apt to be present during the attack, and the pulse is accelerated. The persistent factor is the increased excitability of the motor and sensory nerves to physical stimuli, including electricity, both galvanic and faradic, and upon this fact, in various manner, are based the diagnostic signs of the presence of the disease. Wherever a motor nerve is accessible to pressure, the peculiar spasmodic contraction can be set up in a muscle if it is suddenly compressed or percussed. Trousseau's sign is the most certainly diagnostic of all: compress the biceps, or immediately below the inferior insertion of the deltoïd, and in the lower extremity upon the internal surface of the thigh, and a contraction of the corresponding muscles will ensue. It was supposed to produce its effect by affecting the blood-supply, but it usually does so by irritation of the motor nerve to the muscle. When we percuss the facial nerve at its exit, just below the malar bone, a facial spasm will result. This is Chvostek's sign, but it is not so constant. Hoffman's sign: this sign is present when pressure upon a sensory nerve produces pain or tingling in the distribution of the nerve. This is also present in some, but not all cases, and not always in the same case. Erb's sign: the electrical excitability of nerves is greatly increased, so that a milder current than normal will set up contraction. Both currents are potent, but the galvanic more often than the faradic. The response is practically the reaction of degeneration, i. e., the anodal closure contracture is equal to, or greater than cathodal contracture, etc. The most marked phenomena, however, is the fact that while contraction is not instantaneous, relaxation is greatly delayed, so that the muscle may remain in contraction until the current is cut off. Erb states that all nerves are not equally responsive, the ulnar being most active. Dana says that while the auditory nerve in health is not excitable, in more than fifteen per cent of cases, it always responds in tetany. An anodal opening contraction is found only in this disease.

In accordance with the discovery of conditions in the anterior horn of the spinal cord which would suggest poliomyelitis, we are not surprised to find in some cases vasomotor and trophic symptoms, sweating, herpes, loss of the finger-nails, and muscular atrophy, and that during the attack the face is flushed, the extremities are cyanosed, and the hands are red, which symptoms may pass away after the attack. The reflexes are usually normal, but an increase or diminution is sometimes seen. The objective sensations are not modified, except when hysteria is associated. Indicanuria is so frequent in children that it may be considered pathognomonic. Glycosuria has also been observed. Occasionally epilepsy, exophthalmic goitre, and myxedema have been found associated with tetany.

PROGNOSIS. Epidemic cases are likely to have frequent attacks. These are the febrile cases, but usually they recover within a few weeks, and that is generally the rule in cases where attacks are frequent. When the attacks come on at longer intervals, the duration of the disease is apt to be a number of months. On the other hand, the entire picture may consist of only a few attacks of muscular contraction. Recurrences are not infrequent, and in some cases the attacks recur at regular intervals for a number of years. A case cannot be considered cured until both Trousseau's and Chvostek's sign disappear. Most cases recover, and the usual course of the disease may be considered to be from two or three weeks to as many months, but some cases are fatal, and it is said that those with gastro-dilatation are cured only by gastro-enterostomy, and that the presence of exophthalmos is a very unfavorable symptoms; also, when the respiratory muscles are affected, life is threatened, children particularly being in danger of suffocation.

DIAGNOSIS. The diagnosis of a typical case is usually easy. The disease is peculiar in being a tonic bilateral spasm, affecting primarily the extremities, with a hyper-excitability of the muscles and nerves, and in the presence of Trousseau's sign, which is not found in any other disease. The sensory irritability is rare in any other disease, and the constant irritability of the auditory nerve is also diagnostic.

DIFFERENTIAL DIAGNOSIS. Tetanus differs in the fact that the spasm begins in the muscles of the jaws and neck, instead of in the extremities. In tetanus, also, the temperature is elevated from the outset, while fever is absent in tetany, or occurs only late in the disease, or in epidemic cases. The contractions in tetany are milder, and intermit, and trismus rarely occurs, and if it does, it is late in the disease.

A pseudo-tetany in children, which has a bad prognosis, is characterized by generalized contractions, with albumin in the urine.

In hysteria there is a pseudo-tetany, with tonic muscular contractions, which are unilateral, and painless, and neither Erb's nor Trousseau's sign is present.

Latent cases have been described without spasms, the only symptom being Chvostek's and Erb's signs, with some tingling of the hands.

TREATMENT. Remove the cause if possible. If it is from the strain of lactation, the child should be weaned at once; if during gestation, it is not considered to be cause for abortion, but for treatment. Gastro-intestinal disease is often the cause, and always a factor, and therefore lavage of the stomach, and a thorough cleansing of the bowels is imperative. If any form of intoxication can be discovered, its entrance must be stopped, and in addition to the above-mentioned measures, intra-venous use of normal salt solution should be considered. If it has followed the too complete removal of the thyroid group of glands, the gland substance, or extracts of it should be administered. The parathyroid glands control calcium metabolism, and in case of their inefficiency there is a great increase in the excretion of this salt. It has been proved clinically and experimentally that in the presence of tetany there is a reduction in the calcium salts to about one-half the normal. This is the reason why lactation is so provocative of the disease, and also why a milk diet is often so beneficial. This loss has been supplied with benefit by the administration of the lactate of calcium intravenously. Forty to eighty grains of calcium lactate are added to four or five hundred c.c. of normal salt solution, and injected, and this may be repeated in twenty-four hours. Thyroid gland or extract, or parathyroids, or a nucleo-proteid have all been given, but reports as to their efficiency are not unanimous. These substances have been given by the mouth, and intravenously, the latter method being considered the more efficient. The gland tissue has been placed in the tibia of one patient, and in the peritoneal space of another, and with good results in both cases. When exposure to cold has been the cause, warm baths and diaphoretics are used. In rachitic infants appropriate treatment must be instituted for the basic condition. In conditions of malnutrition, abundant food, rich in nourishment, small in bulk, and in liquid form, is demanded. A milk diet has answered these conditions in some cases. In the gastric tetany of children, gastro-enterostomy is the treatment of choice. In case of asphyxia, due to spasm of the glottis, injections of pilocarpine, cold cloths to the throat, or a hot sponge is advised by another. Tracheotomy may be demanded. Trousseau advised, for treatment of the general condition, ice to the spine; and tepid baths of fifteen minutes' duration, several times a day, have been helpful. Galvanism may be used, but the current must be weak. The convulsions must be relieved, and the employment of Morphine, Bromides, and Chloral are necessary at times. Halbert advises 1/8 of a grain extract of Cannabis Indica. Aconite. Secale, and Solanum nigrum were advised by Trousseau for the different phases of the complaints. Bartlett advises Arsenicum, Cinchona, Strychnia, and Ferrum for the exhausted and malnutrition cases, and for the rachitic infant, Phosphorus, given for a long period. Generally after the convulsions give Phosphorus, Phosphoric acid, and Nux vomica.

MYOTONIA CONGENITA.

THOMSEN'S DISEASE.

DEFINITION. This is a rare disease of adolescence, characterized by hypertrophy of the voluntary muscles, which is not followed by atrophy. In some way the disease changes the muscles so that they are thrown into spasmodic contraction on volitional movement.

THE SYMPTOMS OF A TYPICAL CASE are hypertrophy of certain muscles or muscle-groups, with spasticity on volitional movement, principally noted in the muscles of the lower limbs. The whole body is thrown into contraction, but if it is possible to repeat the movements in spite of the contraction, it finally gives way, and the muscles move normally until after another period of rest.

DIFFERENTIAL DIAGNOSIS. It is to be distinguished from Tetany, Paramyotonia congenita, and Pseudo-hypertrophic Paralysis.

SEX. Many more males than females.

AGE. While cases ordinarily present themselves to the physician in a period from the seventeenth to the twentieth year, careful examination of the history generally reveals the fact that traces of the disease have been evident from the earliest childhood. It has been seen to occur in a man of twenty-five years of age, but is most apt to become conspicuous at about the age of puberty.

HEREDITY. It is practically always hereditary. It is generally familial. Twenty cases have been seen in four generations, and usually there will be found to be several cases in the same family, even if heredity is not traceable. It could be traced in Thomsen's family for five generations.

ETIOLOGY. This is yet in doubt. Thomsen considered it psychopathic, because mental excitement so definitely aggravated the spasm. Johnson and Marshall, from their investigations in the Neurological Department of Guy's Hospital in London, considered the muscular changes as a result, and not a cause. They found that a muscle that responded to a volitional impulse by spasm, responded normally to reflex stimulation. They offer the hypothesis that there is an increase of resistance in the synapse, and that on a repetition of the stimulus the blocking becomes less and less, and that the response to a reflex stimulation is normal, because the higher motor paths are not involved. It has been demonstrated that in some cases the motor end-plates are degenerated. From this fact it has been deduced that an intoxication is the original cause of the degeneration of the peripheral nervous mechanism, and then muscular hypertrophy ensues. The changes in the muscle are so constant that it is far more probable that it is a congenital developemental fault.

PATHOLOGY. There is a multiplication of fibrillæ in the fibres, and the nuclei are in proportion to the size of the fibres. The fibres change from polygonal to a circular shape, and increase in width from their normal limits of from 1/1000th, to 1/400th of an inch to 1/500th, to 1/150th of an inch. Striation, either longitudinal or transverse, shows no change from the normal.

SYMPTOMATOLOGY. The particular and diagnostic symptom is this: when voluntary contraction is set up in a muscle it practically amounts to a tonic spasm, which will not relax for from ten to twenty-five seconds. This is on the initial contraction of the muscle. but, rarely, the second tonic spasm is greater than the initial one. The effect of this condition is, that after a person has been at rest and attempts any motion, these muscles become rigid, and a glass grasped by the hand is held there, further motion or relaxation being impossible. In generalized motions, like an attempt to walk, the whole body may be thrown into tonic spasm, and from the fact that we retain our equilibrium by innumerable compensatory contractions of antagonistic muscles, the patient may fall to the ground, incapable of any motion whatever. This condition of tonic spasm may be generalized, but as a rule, it is more strongly marked on one side than the other. The muscles of the face are usually free, but those of mastication are not uncommonly affected, and in some cases it is impossible to protrude the tongue, swallow, or speak, until, by repeated trials, the spasm becomes relaxed. Sometimes the eyelids show the same spasticity. The finer muscles of the hand are almost always spared, so that while the patient cannot execute forcible movements of the hand with success, he can write with ease. This condition is most apparent after a long period of rest, after chilling, a cold bath, sometimes mental shock. It is minimized by continued exertion, by warmth, and, some have found, by moderate indulgence in alcohol. Erb stated that electrical contractility is changed. There is the reaction of degeneration to galvanism, and the response to faradism is a slow and long-lasting contraction. There is a long, undulating wave starting from the cathode, and going to the anode. The diagnostic point is the sluggishness of the reaction, the tonic character of the muscular contractions, and their long duration. If a strong faradic current is passed through a muscle, it contracts into rigidity, which lasts for twenty or more seconds, and if the muscle is sharply percussed, or even rigidly compressed, a knob-like swelling remains for some time. Respiration, defecation, micturition, or even coitus, may be embarrassed by the spasm. No sensory or mental change.

COURSE. This is slow, and the advent is hardly noticed, but is likely to become more evident about the time of puberty.

PROGNOSIS. Usually incurable, but there may be remissions, and one female case was better after marriage. It does not tend to death.

DIAGNOSIS. This rests upon the presence of an irregularly distributed hypertrophy of muscles, with tonic spasm on attempts at voluntary motion, decrease of this spasm upon continuance of the motions, long-lasting tonic contraction of a muscle after faradic stimulation, or from a blow, or from strong pressure.

DIFFERENTIAL DIAGNOSIS. Tetany occurs in those who are rachitic, the spasms begin in the hands, and Trousseau's sign is found in this disease alone. The sign is this, that pressure upon the brachial artery will produce a spasm in the arms. This disease has premonitory symptoms, which are lacking in Myotonia congenita. They are numbness, prickling, and some local pain before the rigidity, and, in adults, some headache, and feeling of malaise.

Para-myotonia Congenita arises from the effects of cold, which causes a numbness and stiffness. This passes away, but some stiffness remains, and a degree of paralysis. The orbicularis palpebrarum and oris are especially involved. Muscular irritability is not increased.

Pseudo-hypertrophic Paralysis has peculiar deformities, contractures, and weakness. There is no tonic spasm on voluntary motion, nor on faradic, or physical stimulation of the muscles. At a later date it also shows atrophies.

TREATMENT. There is very little ground for hope of relief from remedies, but small doses of Veratrine are quoted by Jolly as beneficial. Others advocate massage, and educational forms of gymnastics. The individual must learn what will aggravate, and what will mitigate the spasm in his own particular case, but these measures are hygienic rather than medicinal. Regular and systematic exercise is advisable in all cases. Strychnia, Cicuta, Agaricus, and Cuprum should be studied, and might relieve.

MYOTONIA ATROPHICA.

DEFINITION. This is a rare and slowly progressive disease, sometimes referred to as Thomsen's Disease with Muscular Atrophy, or some other modification of that symptom-complex. Its peculiarity is a spasmodic condition of some muscles, while other muscles are the site of a flaccid paralysis, the distribution being after a constant order.

HEREDITY. It seems to have a hereditary tendency, as about half the cases can be shown to be familial.

AGE. It is a disease usually of early to middle life; 15 to 30 years.

SEX. Males predominate.

ETIOLOGY. It is too rare to afford more than a hint at its causation, but the infections have been alleged to be causative, yet a developemental fault is probably necessary for their efficiency.

PATHOLOGY. In a case reported by Kennedy there were no pathological changes in the blood or urine, and the Wassermann of both blood and cerebro-spinal fluid was negative. In the muscles the condition is like Thomsen's Disease, but in addition Stewart found degeneration in the posterior columns.

SYMPTOMATOLOGY. The atrophy of the facial muscles is characteristic; with them is atrophy of the sterno-cleido-mastoids, the vasti of the thighs, and the extensors of the feet, and less often, the extensors and flexors of the forearm, the masseter, and the temporal muscles. Kennedy, in his case (Journal of the Amer. Med. As'n., Nov. 29th, 1913) found that after no pathological history except pains in the lumbar region for two years, his patient showed double ptosis, weakness of the orbicular muscles, atrophy of the temporals and masseters, sterno-cleido-mastoids, and some atrophy of the extensor muscles of the forearm. The vasti internus and externus were atrophied, sparing the rectus femoris; there was an almost complete atrophy of the anterior tibial and peroneal muscles, with complete bilateral foot-drop, and steppage gait. Right knee-jerk was difficult, ankle-jerk, and plantar-jerk were absent; abdominal and arm reflexes were normal. There was bilateral cataract.

The myoclonus spasm is seen only in the first five or six grasping movements of the hands. The stronger the contraction the longer it requires to relax it. The electrical reactions are like those of Thomsen's Disease, i. e., easy exhaustibility.

DIAGNOSIS rests upon a myotonus of the arm, combined with the atrophy of the face and neck muscles constantly, and of the forearm, thighs, and lower limbs less constantly.

PROGNOSIS. It is slowly progressive, and terminates in death.

TREATMENT. There is much doubt of the efficiency of any treatment, but a free feeding of fats, as in the atrophies, would be a rational basis.

Cicuta, Agaricus, Strychnia sulph.. or perhaps better, Strychnia phos., with Cuprum might be of value.

FAMILY PERIODIC PARALYSIS.

DEFINITION. It is a form of flaccid paralysis which comes on while the patient is presumably in a state of perfect health. There is no loss of consciousness; its duration is from a few minutes to a week; it recurs every few months initially, but when established may come on with varying intensity every few days; it may attack several members of a family, and it can be traced for several generations.

ETIOLOGY. The fact that it is hereditary, descending through both sexes, stamps it as the result of some inherent instability of nervous or muscular tissue, but nothing more is known. The exciting cause is equally in doubt, but cases have seemed to be badly influenced by fatigue, and indigestion, and therefore it is probable that there is a deficient elimination of toxins. This is also indicated by the fact that the attacks have been lessened by increasing elimination, and also by the administration of sedatives. Attacks generally begin early in life, before the age of twenty-five, but rarely before the tenth year. Examination of the muscles, and its hereditary quality has suggested community with the atrophies and dystrophies, while its manner of onset suggests an intoxicant, and allies it with epilepsy.

SYMPTOMATOLOGY. Attacks may be mild or severe, but are generally ushered in by tingling sensations, or even pain in muscles. The patient may, and perhaps most often does, awaken out of sleep in a condition of paralysis. Usually the paresthesias are followed by a sensation of intense weariness, and the loss of power in the lower limbs, rapidly rising to the trunk, and then involving all the muscles of the body, with the exception of those innervated by the cranial nerves. The bowels and bladder during the attack may be incompetent, but the sphincters are intact. This order of involvement is not universal, and is singularly like Landray's Paralysis. Some cases are reported where deglutition was slow, and the voice was weak. The paralysis is flaccid, the reflexes are abolished, and electrical reactions are greatly reduced, or absent entirely. Attacks may be mild or severe, and therefore in some cases the person feels only an excessive weakness, while in others they are entirely helpless, even unable to lift the head from the pillow. Sweating is a very frequent symptom. Priapism and cardiac weakness were symptoms in one of Starr's cases.

Prognosis. Absolutely good for recovery of muscular power after an attack. The attacks tend to grow lighter late in life, but instances of death are not unknown.

Treatment. This has been directed toward an increase in elimination, sedation, and an improvement in the diet between attacks. Gelsemium is the remedy which suggests itself before all others.

CHAPTER XVI.

NEURASTHENIA.

DEFINITION. This is a condition of irritable weakness of the nervous system, and as a result there is an inordinate response to external stimuli, and an amount of subsequent fatigue out of proportion to the exertion, whether it has been mental or physical.

DIFFERENTIAL DIAGNOSIS. It must be differentiated from Anemia, Chronic Alcoholism, Exophthalmic Goitre, Hypochondriasis, Hysteria, Lithemia, Epilepsy of the Petit Mal type, General Paresis, Disseminated Sclerosis, Cerebral Tumor, Cerebral or Cerebro-spinal Syphilis.

AGE. No age is exempt, but young adults furnish most of the patients.

SEX. Pure forms are more common in the male.

ETIOLOGY. Heredity is the most important factor, and alcoholism in the parent is one of the most common causes of such a neuropathic constitution. Another frequent cause is tuberculosis in a parent, insanity in another, and an anemic condition of the mother at the time of pregnancy, either from malnutrition, or from too rapid child-bearing. A sedentary life with a monotonous occupation of a mental kind, involving long periods of continuous application, or general malnutrition from hurried, or scanty, or imperfectly cooked meals may give rise to it. Defective teeth may produce the condition by the imperfection of mastication, or from the absorption of the toxins from the dental disease. Occupation involving night-work, since the sleep in the day is liable to be abbreviated in amount, and is not so restful in quality as that taken at night. The above are predisposing causes.

Exciting causes are numerous, but worry is the most common and most efficient; it is the modern straining of one's powers in the race for wealth and position. Emotional shocks, whether involving what we term fright, or whether it be impending disgrace and humiliation. Traumatism even without any discoverable lesion may cause it. Alcoholism, especially if combined with excessive sexual indulgence, and the various forms of such excess, masturbation, and interrupted coitus, are competent to produce it. Any chronic physical derangement which involves distress or discomfort may produce it in a neuropathic individual, and hence we see it following uterine disorders, neuralgias, adenoids and eyestrain. It is often toxic from alcohol, morphine, cocaine especially, and also from lead and arsenic, and certainly sometimes from tobacco. It is very often toxemic; directly from influenza, and indirectly from carcinoma, tuberculosis and like conditions. It is thought by some to be the result of auto-intoxication, since a large proportion of cases is preceded by chronic gastro-intestinal indigestion. The early stages of Graves' Disease, Hodgkin's Disease, Addison's Disease, and Raynaud's Disease may be attended by it. Indigestion may be a symptom proceeding from the neurasthenia, but in such case, it will be found to bear no fixed relation to meals; it is variable in extent from day to day; it will occur at times after digestible food, and be relieved by that which is undigestible; it is always accompanied by other neurasthenic symptoms, and the attacks are not amenable to the ordinary remedies for such conditions. Far more commonly we find an indigestion with a normal symptomatology. It has a history long antecedent to the neurasthenia; as this indigestion improves the neurasthenia is benefitted, and if a relapse in the gastric condition takes place the neurasthenia will be proportionately increased. If the gastric condition is chronic, we are apt to find an exacerbation of nervous symptoms at a definite, fixed period after meals, seeming to indicate that a toxic material is poured into the blood at intervals.

When we find cases of generalized disease arising from an evident intoxication, we always find the patient exhibiting headache, insomnia, restlessness (both mental and physical), nervousness, prostration, hyperesthesias, pains, irritability of temper, readiness in being startled, easy fatigue, deficient memory, and a deficiency of the power of attention. These are the symptoms also of neurasthenia, and lend great probability to the hypothesis that, however initiated, the condition actually arises from the action of toxins introduced from without, or generated within the body, generally acting in combination with a neuropathic heredity.

PATHOLOGY. While no lesions have been demonstrated, it is most probable that it is a morbid entity developed as an exhaustion of nerve-cells. Cases differ sufficiently in symptomatology to be spoken of as cerebral and spinal, but such a localization is not exact. The easy fatigue of the mind, the defective memory, and the reduction of the power of attention, show that the cortical cells presiding over intellectual processes are the site of an exhaustion process. The slowness of motor response on the one hand, and the muscular jerkings and twitchings on the other, point to a weakened and irritable condition of the cells in the motor part of the cortex. These muscular starts and jerks may in other cases have their source in the pathological condition of the cells of the cord, and of the muscles themselves. These symptoms exist to some extent in almost every case, but they are not so persistent as the flushings, palpitations, bursting sensations, sometimes the actual swelling of a part in limbs and body. We know that such symptoms can result only from disturbances of circulation, and that the calibre of arteries is controlled by the sympathetic nervous system. It presides also over the secretions and the functions of the viscera, and would explain the imperfection of digestion, and the vagaries of excretion, which are so prominent a feature of this disease. It would probably most accurately localize the lesion if we ascribed the condition to a toxic infection of the general nervous system, particularly evident in the domain of the sympathetic. The reeling, swaying, dizziness, vertigo, agoraphobia, and claustrophobia. for instance, result from a disturbance of the circulation in the semi-circular canals, and the contiguous structures.

SYMPTOMATOLOGY. The symptoms may be summarized thus. Easy fatigue, irritable weakness, depression, indefinite fear, indecision, disinclination to mental or physical exertion, mental capacity unimpaired in quality, but easy fatigue makes it inefficient. Headache, vertigo, insomnia, bodily restlessness, motor weakness and tremor, and the deep reflexes are exaggerated. The headache may be only a feeling of heaviness, or, on the contrary, of lightness or emptiness of the head, but typically it is a splitting weight upon the brain, with a tightness of the scalp, which has been so often found that Charcot termed it the "casque neurasthenique." The neurasthenic source of this pain may be recognized from the fact that it increases on attention, and is localized according to the conception of the supposed causative disease. Hemicrania may occur. Vertigo is usually without the motor incoördination which might be expected if it were organic. The patients protest that they will fall if they attempt to walk, but can actually walk to the extent of their muscular power.

Insomnia is usual, and is persistent, and sleep is disturbed by vivid dreams, and by muscular starts and twitchings. The patients are sleepy, and the dreams are worse in the half-awakened state in early morning.

The Eyes show a contraction of the visual fields after use (a fatigue symptom), and one patient could not look at pictures in a gallery, since the image of the first one persisted, and blurred that of the next. Muscæ volitantes and photophobia are frequent, and the eyes develope latent muscular defects. There are ringings and buzzing noises in the ears, and a very pronounced over-sensitiveness to noises. There may occasionally be a neurotic impairment of hearing. These senses show no signs of being diseased, however, but are too easily fatigued.

Gastric symptoms are almost always present. Collections of gas are common and distressing, and there are acid eructations and distress from food, generally irrespective of its character.

Constipation is present, and the patient passes large quantities of urine. Owing to the large discharge of phosphatic salts (oxalates, Oppenheim) there is apt to be some cystitis, and occasionally there are pains at the neck of the bladder, simulating the crises of tabes.

Pains are a prominent symptom, and, after the headache, the most common site is in the spine and the limbs. The area between the shoulder-blades, and at the nape of the neck, are points of election. They may be about the waist, and one patient complained of them at the "back of the stomach." The pains in the back were formerly called "spinal irritation," and "rachialgia." Sometimes one particular spot is affected, and it is accompanied by a tenderness over the corresponding spinous processes. Fixed pain is common, but it is generally in the muscles and ligaments of the back, from lack of muscular tone, and is such as one suffers from sitting, or standing, for a long time in one position. There is a weariness like that resulting from unaccustomed exercise. Tenderness of the spinous processes to percussion is not a characteristic symptom. While the patient may complain of severe pain, the facial expression does not confirm the statement.

Bodily restlessness with weakness is present in every case, and it even extends to inability to be contented in any place. Examination will show that every kind of movement can be executed with precision, and that there is no atrophy nor reaction of degeneration, but movements are initiated slowly, there is little force to muscular contractions, and fatigue is rapid.

Since a certain amount of hypochondriasis always accompanies neurasthenia, the weakness suggests the advent of paralysis to the patient. We can assure them that the hypothesis is fallacious, since the weakness is never confined to one nerve area, but is always general; it is unaccompanied by atrophy, and there is no reaction of degeneration.

Tremor is usually present, and is fine (may be fibrillary), and rapid. It is exaggerated by exertion, exposure to cold, and by emotional strain. Shaking fits may occur.

The Deep Reflexes (particularly the knee-jerk) are exaggerated, and we may find a foot-clonus, but this is exhausted after six or seven oscillations, thus showing that it is not organic. Rarely the knee-jerk may be very much reduced (only from extreme muscular emaciation), but if it cannot be elicited by some of the methods of re-enforcement it is organic. The superficial reflexes are greatly exaggerated, and the exaggeration of the plantar reflex may give rise to excessive jerking of the legs, and pain in the back. There is no relation between the grade of the reflex-response and the gravity of the disease.

Changes of Sensation in the skin are common, but while paresthesias, formication, a feeling of coldness, pruritus (particularly of the anus) with tic-like twitchings, are common, anesthesias are rare. The nearest approach to anesthesia is a feeling of coldness and deadness in the extremities which is very common. This is vasomotor. Alopecia is a common symptom.

The Circulation is always disturbed, and palpitation and tachycardia are present in some stage of all cases. There may be bradycardia, and a lack of coördination in the contractions. The attacks may be so severe as to simulate angina pectoris. The pulse rate may go as high as two hundred to the minute. Heart murmurs may occur, but are rare, with or without a disturbance of rhythm, the whole surface of the body may flush irregularly, mostly from emotional excitement. Bursting sensations, and actual swellings of the limbs may occur. Slight irritation of the skin may so excite it that urticaria results. There may be sweating, local or general.

Sexually the patient is weakened, and he may have spermatorrhea, or the parts may be so erethistic that masturbation is induced. Erections are another annoying result, and patients are often impotent. This is actually a hypochondriac symptom, and often has its source rather in a doubt as to their ability, than in any physical weakness. In the sexual cases, nervous cardiac symptoms, vasomotor phenomena, and nervous dyspepsia are prominent.

Mentally the patient is distressed by delusions and hallucinations. He lives in an atmosphere of fear, of which dread of disease is the most common. He fears to be left alone, he fears contracted surroundings (claustrophobia), and the sight of wide spaces terrifies him (agoraphobia). These persons have a fear of the obsessions which impel them to murder or suicide. Sudden noises terrifying them almost without exception. The mind, like the body, is easily fatigued, and there is a deficiency in spontaneous thought. Inability to make, or to hold to decisions is marked. It always tends to be tinctured with or to shade into hypochondriasis.

COURSE. It is generally years in its evolution, although some particular strain, which brings the condition to a climax, when it is no longer negligible, is often considered to be its sole cause. It generally lasts for some years, but may be marked by remissions. It always tends toward hypochondriasis.

PROGNOSIS. Cases are generally curable, more often in the male than in the female, since the female often becomes a patient from vacuity of life, which is a condition which we cannot always remedy. With a bad heredity, the prognosis is doubtful, and also if the patient is degenerate, relapse will be common. The stronger the patient, and the more congenial the normal life, by so much is the prognosis improved. It does not tend to termination in any one of the various forms of insanity. Severe cardiac symptoms may end in atheroma. Traumatic cases tend to recovery, but it may be delayed for years.

DIAGNOSIS. This rests upon the presence of irritable weakness, both physical and mental, an increase in reflex irritability, cardiac and circulatory disturbance, secretory perversions, and tremor either spontaneous, or readily excitable.

Differential Diagnosis. Hysteria. Both this and neurasthenia may be initiated by shock, and there may be an interval of weeks between the traumatism and the outbreak. Although both patients may start at slight noises, have depression, palpitation and pain in the head, at the top, and insomnia, the history of transient and anomalous nervous attacks, intermittency of many of the symptoms, and ovarian tender spots, stamps the case as one of hysteria. Neurasthenia is marked by more or less lasting exhaustion, hysteria by the occurrence of the phenomena in paroxysms. Cases are often anomalous, and difficult of identification. Hysteria has deficient will control, and an increase of reflex excitability. Contracted visual fields are found in neurasthenia less often, than in hysteria, but dyschromatopsia (vagaries of color-fields) are found only in hysteria.

Hypochondria. Neurasthenia is an exhaustion of the nervous system, and the mind shows easy exhaustibility. In hypochondriasis there is no mental feebleness, but instead an exaggerated introspection.

Lithemia. This shows a deposit of lithates in the urine, the patient has mental hebetude instead of irritability, vasomotor phenomena are absent, as are other nervous phenomena; headache is severer, but shorter in duration, patients are somnolent instead of having insomnia, but the two maladies are sometimes difficult of differentiation.

Exophthalmic Goitre. Before the eyes protrude, or the thyroid swells, it cannot be distinguished with certainty, but if the palpitation and cardiac symptoms are very prominent, and resist treatment, exophthalmic goitre should be suspected.

Chronic Alcoholism. This has tremulousness, fleeting pains in the limbs, and debility; but the feces are distinctive, the liver is disordered, and the history should be a guide.

Senile, and Other Tremors. Should be easily differentiated.

Malarial Poisoning and Anemic Conditions. These have characteristic signs, physical symptoms and history, and the same is true of Syphilis, Latent Carcinoma, Latent Tubercle, Addison's Disease, Hodgkin's Disease, etc.

Petit Mal and Migraine. Some cases have a paroxysmal quality, reminding one of petit mal, migraine, and other paroxysmal disorders. Here the physical signs are generally wanting, but petit mal has an aura, migraine scintillating scotomata, and in both patients are well between the attacks.

Treatment. This condition is an end-product, and no specific is known, nor is it a case for specific therapeutics. The patient must be put into the best environment, his life regulated to the minutest particular, and his general condition improved in every possible way. Time is an essential element, and must be liberal in amount. If the case is toxic, the dyspepsia and constipation must be relieved in order to stop a constant reinfection. All processes from mastication to defecation, should be put in the best condition. Diet, method of taking food, its quantity and the interval between meals, should be specifically stated. Enteroptosis should not be forgotten, as a patient of my own began to improve only after a proper abdominal compress had been provided. Make a thorough physical examination, since the anemia of lasting constitutional disease often masquerades as neurasthenia. Galvanization of the cervical sympathetics and along the pneumogastrics frequently helps digestion. One of the best methods of freeing the bowels is by means of large enemata of warm water to which a little boric acid has been added. The best soapsuds injection is made extemporaneously by mixing two tablespoonfuls of soda bicarbonate with the same quantity of olive oil in a little water, and administer, with the patient lying upon the back.

Rest is next in importance. Use rest, but do not bar cheerful society. Isolation is not needed as it is in hysteria. Hypnotics for sleep: 12 hours sleep in the 24 hours. Take breakfast in bed. Faradism general, and the D'Arsonval high-frequency current. For the nervous irritability use hot or Turkish baths. Spinal weakness will be benefitted by cold douches to the spine. It needs several weeks of baths to obtain their value. Gentle muscular exercise is good. It benefits cerebral neurasthenics, but hurts the spinal cases. A sea voyage is good "per se," and it also removes one from the possibility of injury from the routine of daily life. The moderate use of tobacco may soothe the unusual patient. One cerebral case characterized by restlessness, was observed to surreptitiously inhale the smoke from her husband's after-dinner cigar, and was noticeably better after it. She was urged to follow his example, and under the influence of one strong cigar daily made a prompt recovery.

Electrical Treatment. In approaching this form of therapy we must keep in mind that cases fall into three pretty clearly defined classes, viz., the cerebral, the spinal, and the cerebro-spinal. In the mild cases no accurate classification is demanded for electrical treatment. They will all be benefitted by a general faradic massage for ten minutes at the termination of the usual rubbing. While local galvanic treatment through the brain may be demanded in some cases, the equalization of the general circulation by general faradization has seemed to fill all the indications in the author's practice. In these cases the static machine has been of great service. Place the patient in the circuit, with the feet upon the plate, and discharge through the hair by the use of the crown. The most noticeable effect has been a sense of mental well-being and an improvement in the circulation of the extremities. This treatment should be maintained for from ten to twenty minutes. The spinal cases have seemed to be least amenable to treatment, but the static sparks from the spine have relieved some of them. In the cerebro-spinal cases the galvanic treatment of the cranium should be supplemented by sparks drawn from the spine, and the static breeze applied to the spine. The insomnia should always be treated by general faradism if it is well borne, but it should be kept in mind that it disagrees with, and aggravates some cases. High-frequency currents have been extensively used in neurasthenia. They are said to be contraindicated in restlessness and insomnia, and are suitable only for the cases which are relaxed, and who complain of complete lassitude. Auto-condensation with the effluve or local glass electrode along the spine has been said to be useful in such cases. The author's personal experience has been that while auto-condensation has produced striking changes in temperature and pulse rate, it has not produced much effect upon the neurasthenic condition. On the other hand, if the effluve, or glass electrode, has been at the same time applied to the organ, or part, most distinctly the seat of the symptoms, the function of these parts has been very appreciably benefitted. The liver and kidneys are especially improved, but treatments applied to the spine for the relief of pains, either local, or in the distribution of the nerves from definite segments, have not been of any appreciable benefit.

THERAPEUTICS. Abies nigra. For atonic stomach, with depression, and mental dulness.

Aconite. Erections and nocturnal emissions, without voluptuous dreams. Eructations and inclination to vomit.

Æthusa cynapium. Inability to think, or to fix the attention. Stomach and abdominal pains are distinguished by their violence. Irritability, especially in the open air.

Agaricus musc. Numbness of occiput and spine, with muscular twitchings, and mental confusion (Stephenson).

Alumina. Restlessness and mental exaltation with general fatigue, and a constipation characterized by an inability to pass even a soft stool. Must be forced out.

Ambra grisea. Nervousness and bashfulness with forgetfulness, easy fainting, and a voluptuous itching of the pudenda. Pelvic tenderness.

Ammonium mur. Coldness and sensitiveness to cold, with a fear of darkness. The head feels full, with weight on the forehead.

Ammonium picrate. Congestive symptoms in brain and cord. Heavy feelings in occiput and mastoid region. Wild feeling in occiput following traumatism.

Amylinum nit. Flushings of face, and bursting feelings over chest. Constriction of heart and throat.

Anacardium orientale. Nerve exhaustion and nervousness, fixed ideas, obsessions, girdle sensations. Sense of complete exhaustion.

Anhalonium Lewinii. This has symptoms of mental hebetude, tired feelings in the brain, as well as the muscles; vertigo and faintness; muscæ volitantes, and visual hallucinations, due to a combination of cerebral and ocular disturbance. All the senses are very acute.

Argentum nit. Headache and dyspeptic symptoms from mental overwork.

Arsenicum alb. Restless weakness with irritability. Pronounced gastric irritability.

Artemisia absinthium. Nervous, restless weakness with vertigo.

Asafœtida. Great flatulence with watery stools. Great sensitiveness to physical contact, and the pains are characterized by numbness of the affected parts.

Asarum Eur. Exceeding over-sensitiveness to slight noises, with nervous irritability, and a sense of levitation.

Asclepias Syriaca. Polyuria of neurasthenia. Belching of food unchanged by digestion. Vertigo, dullness, and stupidity with a sense of constriction across the forehead.

Asclepias tuberosa. Similar, but with much vertigo.

Asterias rubens. An apprehensive state of mind with alternations of sorrow and mirth. Vertigo is prominent. There is constipation. There are nocturnal pains in the breasts, and a pushing out sensation in the uterus. The face is red and congested.

Athamanta. A confusion of the brain, indigestion, and an icy coldness of the extremities.

Aurum met. Useful in young patients with night terrors, which are a form of neurasthenia.

Avena sativa. In doses of 5 to 15 drops of the tincture in hot water, a remedy in general.

Badiaga. General tremor with palpitation.

Baryta carb. Motor and mental asthenia.

Belladonna. With characteristic symptoms, and a peculiarly burning feeling as if in the brain.

Bellis perennis. Vertigo, easy fatigue, feeling of contraction in the scalp, "casque neurasthenique."

Cactus grand. For cases with much precordial distress.

Calcarea carb. Night terrors in children.

Camphora. A feeling of utter weakness, with a conspicuous coldness of the surface, and particularly of the extremities. Insomnia, and feeble action of the heart. Visions of spirits in sleep.

Camphor brom. 3x. Nervous excitability. Hysterical alternations of mood.

Carbo veg. Flatulence, debility, with vertigo, with an aversion to darkness, and fears of ghosts.

Carduus benedictus. Muscæ volitantes, and the eyeballs feel burning and large. Blackness before the eyes at times.

Castoreum. Nervous women with much debility, and as an accompaniment, pains and cramps in the abdomen.

Chionanthus Virg. A case is reported of the cure, by this drug, of a clergyman, who complained of continual perspiration during the summer, weakness in arms and legs, prostrated after preaching, mind ran on his sermon and kept him awake, stupid and drowsy all the time, appetite poor, and food gave him distress.

China officin. Muscæ volitantes, weakness physical and mental, irritable and despondent.

Cobaltum. Pronounced pain in the back, frequent nocturnal emissions, with lewd dreams, with partial, or absence of erections. Impotence.

Cocculus. Characterized by an empty feeling in the head, an opening and shutting sensation, and a sense of great bodily fatigue. Irritable weakness of all the functions of the body.

Coffea cruda. Nervous over-impressionability.

Coniinum. Fatigue, and particularly the muscular sense of fatigue found after unaccustomed exercise.

Conium. Nocturnal emissions, and ejaculations upon slight stimulation. Girdle sensations.

Curare. Mental and physical lassitude with lancinating headache.

Cyclamen. For depressed cases with vertigo and headache.

Cypripedium. Mental irritability, and excitement from overstrain. Especially in children.

Daphne Indica. Blurring of vision with constipation, and the peculiarity that the tongue is coated on one side only. Headache provoked by intellectual labor.

Datura met. Investigate symptomatology when extreme dilatation of the pupils is a pronounced feature. Obsessions to suicide, and violence to others, with changeable disposition, and exalted ideation with vertigo.

Digitalis. Cardiac weakness, easy fatigue, and emissions in sleep.

Dirca palustris. Vital depression, pains in the head, flatulence, palpitation from slight causes, insomnia.

Eupatorium aromatica. Nervousness, and nervous irritability, with tremors and jerkings of the limbs.

Ferrum phos. Mental hebetude, with irritation at his deficiencies, in combination with the circulatory irritability of the Ferrum group.

Gelsemium. Mental and physical lassitude. Polyuria, diplopia. Muscular tremor. Symptoms in the young especially. Neuralgias in various locations.

Glonoine. Blushing and flushing of the surface with tachycardia and cardiac pain, and hammering headache, intolerance of the heat of the sun. Great sensitiveness of the head to the least jar.

Gnaphalium. Sexual excitement.

Heloderma. Symptoms of cardiac compression, with a general icy coldness, intense in degree.

Hura Braziliensis. Nervous excitability and twitchings, combined with a similar degree of irritation in the gastro-intestinal sphere.

Kali arsenicosum. Morose and depressed condition of mind, and nervousness, constricted form of headache, photophobia, loss of appetite with salivation, burning throughout the whole gastro-intestinal tract, globus and dyspnea, spinal tenderness and general muscular weakness, and cramps.

Kali phos. Despondency, anxiety, fearfulness, tearfulness, homesickness, suspiciousness, agoraphobia, weak memory; small frequent pulse, and later a retarded one, pains with the sensation of paralysis; weakness of muscles even to paralysis, retarded nutrition, depression of function throughout the body.

Lachesis. This is much like the preceding, but with a predilection for the left side in all symptoms, and a remarkable dislike to being touched.

Magnesia carb. There is a typical nervous exhaustion. The keynote is sensitiveness. This relates to mental impressions, touch, cold air, shocks and blows. This hyperesthesia often passes into neuralgias.

Methylene Blue. In the 3x, neuralgias of neurasthenia, tremors and trophic disturbances; in irritations of the spinal cord, and where the patient is very tympanitic.

Onosmodium. Vertigo and general sensations of numbness, and a prostration of muscular strength. Neuralgic pains with this. Numb feelings in the head. Spinal cord irritation, and sexual excitement. Cases which have been developed from sexual excess.

Oxalic acid. Neurasthenic cases with notable cardiac symptoms, palpitation and irregular action alternating with aphonia.

Phosphoric acid. Marked apathy and drowsiness, confused and disconnected thoughts. Mental exertion makes him dizzy, vertigo with the symptom that that upon which he is sitting is rising, unequal dilatation of the pupils, muscular weakness and polyuria.

Picric acid. Easy fatigue, both mental and physical. Mental exertion gives rise to burning pains in the spine, occipital headache. Sexual excitement, but with loss of power. A patient of the author's had emissions on mental exertion. The remedy, with the static breeze, and improved hygiene, relieved the condition.

Platina. Arrogance is the keynote, alternation from joy to terror, spine painful, "casque neurasthenique."

Sabal serrulata. Mental irritability, but with weakness. Sexually excited; polyuria.

Senecio aureus. Globus is a marked symptom, and it is peculiarly, and perhaps only applicable to cases where the condition has arisen from suppressed or deficient menstruation.

Stannum. This has mental and physical weakness as its cardinal symptom. The mental weakness is directly due to cardiac weakness; it is an ischemia. This is proven by the symptom that the weakness is felt more on going down, than in ascending stairs. There is also much palpitation. There is a sinking feeling in the epigastrium, and in the chest.

Strychninum. The peculiarly constricted headache (casque neurasthenique), often found in neurasthenia. Facial neuralgia, and neuralgic headache. Pains and chills in the occiput, and nape of the neck, which run down the spine. Itching over the whole body, and particularly in the roof of the mouth. Jerkings and twitchings of the muscles, and limbs, with a hypersensitiveness to all external stimuli. This last is very pronounced.

Thyroidinum. In the 2x for puffy and obese patients.

Vipera communis. This is almost a specific for the bursting sensations in limbs "if they are allowed to hang down."

Zincum. Cases described as "brain fag." It affects muscles, nerve, and brain. It applies to cases where the neurasthenia is not the expected failure of a congenital neurasthenia, but where the condition is the legitimate result of overstrain from work, or dissipation.

CHAPTER XVII.

OCCUPATION NEUROSES.

CRAFT PALSIES.

DEFINITION. Workers in occupations requiring the continuous repetition for long periods of an identical set of coördinated movements are apt to develope a cramp, tremulousness, incoördination, or paralysis in these groups of muscles. The peculiar disability is not evident in the ordinary use of these muscles, nor in as finely coördinated movements if they are of another variety, but exclusively from that set of movements where the disability first manifested itself. Thus a person who suffers from writers' cramp may be able to play the piano with ease. Such a condition is called an occupation neurosis, and it has been developed in performers on various musical instruments, seamstresses, cigar-makers, gold-beaters, and artistic workers in metals, telegraphers, etc. The cramp of writers is the most common, and is here described as the type form of the malady.

DIFFERENTIAL DIAGNOSIS. Any form of paralysis, tremor, cramp, or ataxia of the arm and hand may suggest it, but the history is usually so clear that there ought to be little ground for doubt, and beyond that, differentiation depends upon the fact that in other cases the symptom is developed by any movement of the muscles, while in writers' cramp it is brought out by the craft-movements exclusively.

ETIOLOGY. It is caused by a too prolonged tonic contraction of certain muscles, or a too frequent repetition of a set of coördinated movements by the same set of muscles. With this, or more essentially possibly, there has been a too continuous drain upon inter-related nerve-nuclei, and the cortical centers for coördinated movements. The basic cause in most cases is a weakness of the nervous system, a neuropathy, which may be hereditary, or will be as efficient a cause if it is the result of dissipation, infection or privation.

PATHOLOGY. In considering the various craft- palsies it must be remembered that in some occupations, requiring fine muscular adjustment, the result is a tonic contraction of some muscles almost without relaxation, and many other muscles approximate to this condition. There is a conspicuous absence of the alternate contraction and relaxation which washes a muscle free of fatigue toxins. The favorable results of some forms of treatment have indicated that the distinctive symptoms have resulted from a fatigue-intoxication of the muscles and nervous structures within them, rather than from an exhaustion of the nervous centers. In many, and perhaps the majority of cases, there is evidence that the nervous centers are at first fatigued, and if the strain is still continued molecular changes ensue in the nerve-cells. In other cases the underlying neurasthenia seems to have indicated that the cells of the cortex have been the point of incidence, and that the condition is a psycho-neurosis. Some cases have their origin in a neuritis, and are properly cases of this disease, and Dana says that there is some degree of neuritis in all. Nodular changes in the muscles, and sclerosis of the vessels have been demonstrated in some cases, but while such changes are competent to produce disabilities of this type, they are not to be considered cases of occupation neurosis.

SYMPTOMATOLOGY. The trouble developes slowly, the patient finding that he tires more easily than usual, that he is clumsy, and that his fingers grow numb and stiff after writing. All these symptoms are relieved by rest, and perhaps a little rubbing of the fingers and hand. As time goes on the fatigue comes on almost as soon as writing is begun, certain muscles cramp up, there may be a definite ataxia, and sometimes pain becomes a feature, but it is not spontaneous, demanding movement of the muscles for its production. Three forms of developement are recognized, viz., Spastic, Paralytic, and Tremulous. They are rarely pure in type, but indicate the condition in a general way. The spastic form is most common, while the tremulous type is the least so. In the spastic form the fingers knot up at the first attempt at writing, but at first only after fatigue has been felt. Later it will be manifested on first grasping the pen. Pain is more apt to complicate this form, not spontaneous as was said before. The paralytic form is shown by a sudden sense of fatigue and exhaustion on the first attempt to write, soon passing away if the attempt is given up. A true paralysis of the abductor of the thumb has been seen. The tremulous form may be an element of the paralytic condition, and be exhibited as a general tremor of the arm as well as the hand on attempting to write, or constitute a warning of the paralytic stage, or it may be in pure form. It is then usually considered to be a psychic symptom appearing as the result of the neurasthenic state of the patient, rather than an expression of a muscular disability. It appears open to question whether the impending loss of earning power may not be a reasonable cause of some of the nervous symptoms which are urged as the essential cause of the malady. The patient endeavors to steady the weak hand with the well one, he obtains a larger penholder, or some one of the devices whereby the whole hand is employed instead of the contracted fingers, but finally writing by any method will probably become impossible. If the other hand is educated to do the work, the same cramp is apt to appear in that member.

DIAGNOSIS. This rests upon the appearance of some of the disabilities described, from some occupation competent to produce them, and essentially that they appear only in the prosecution of that craft. Differentiation depends essentially on the history, and this point of unique appearance. An hysterical person might show a disabling tremor when attempting to write, but it would appear in some other conditions, and the stigmata of the disease may be discovered, or the history would give one a suggestion of the true condition.

PROGNOSIS. The prognosis for the relief of the spasm tremor, or paralysis, is good if the person will refrain from writing. Some cases appear to have been cured, and the person has been able to resume his occupation, but cessation during treatment has generally been necessary, relapses are common, and final disability, so far as writing is concerned, is true of many cases.

TREATMENT. Mild cases may be relieved by a round-arm style of writing and an open position at the desk. In the next place, relief may be obtained by making use of some form of apparatus, out of the many, designed to allow the person the use of the whole hand in writing instead of the fingers. The final attempt at cure is by a total cessation of the occupation which has caused it. In all these plans we may advantageously make use of massage, and exercises designed to strengthen the implicated muscles. Electricity is of doubtful utility, but Dana reports good results from the use of a high tension faradic current with the long coil. He also advises the daily use of the stabile galvanic current of from ten to fifteen amperes, anode over the cervical spine, and the cathode over one affected muscle after the other. Gordon reports a number of cures without interruption of occupation by inducing hyperemia by the Bier method. He compresses the arm above the elbow with a bandage for one hour twice a day. This would indicate that muscle fatigue was an essential etiological factor in some cases. Any underlying neurasthenia should always receive attention.

REMEDIES. The Old School do not claim large results from medication and employ mostly the nerve tonics and sedatives. If the nervous centers are exhausted and organically injured, we can hope for little more from our remedies, but the etiology is not quite as definite as that, and many of our remedies show applicability to the symptoms. Rest, massage, and all reasonable adjuvants must not be overlooked in our therapeusis. Hughes advised Arnica in the cases which we recognize as having a large element of muscular fatigue, or neuritis. Hypericum might be applicable in the latter event. He also reports cure from Gelsemium in the tincture. Dewey adds Argentum met., Ambra grisea, Stannum, and Sulphur. From the pathogenesis one should find possible benefit from Anacardium, Calcarea carb., Camphor, Causticum, Cuprum, Jatropha, Secale, Veratrum alb., and Viburnum opulus.

CHAPTER XVIII.

PARALYSIS AGITANS.

PARKINSON'S DISEASE, SHAKING PALSY.

DEFINITION. This is a disease which has been classed among the neuroses, on account of the lack of any constant pathological findings. It is characterized by a general rigidity of the muscles, with a great deal of apparent weakness. This rigidity distorts the body and the limbs in flexion, and produces the characteristic attitude, and gait. The face loses its expression, and since mental operations are performed as slowly as are the physical movements, the patient appears to have some degree of dementia. The extremities, and perhaps the whole body, are affected by a coarse, passive, rhythmical tremor of moderate rate.

SYMPTOMS OF A TYPICAL CASE. There is an increasing rigidity of the whole body, without an increase in the deep reflexes, a tremor which is coarse, rhythmical, and constant, except in sleep, and is passive in type; the gait is trotting, the face is expressionless, and the speech is high-pitched, and monotonous.

DIFFERENTIAL DIAGNOSIS. It is to be distinguished from Multiple Sclerosis, Simple Tremor, Senile Tremor, General Paresis, Hemiplegic Tremor, Toxic Tremor, Traumatic Neurasthenia, Senile Paraplegia.

AGE. It is a disease of the latter part of life, and the vast majority of cases occur between the age of forty to sixty years. This rule is, however, not an invariable one, since well-known observers have reported cases as young as twelve, and a large number, in the aggregate, before the fortieth year.

SEX. More males are affected than females.

HEREDITY. It is not classed among the hereditary diseases, but while direct heredity is not so common (Bury reports two cases, Borgherini three, Gowers two, Pennato two, and others, here and there, in literature) there is always evidence of a neuropathic condition in the parents.

ETIOLOGY. This depends upon a predisposing neuropathic condition, which may be inherited, or may have been acquired. While it has often been noted that it is a disease which seems to pass the profligate by, and attack the industrious and virtuous, equal stress has not been laid upon the fact that those industrious and virtuous persons have, almost uniformly, so lived and worked as to reduce their vitality, and render the tissues extremely liable to degeneration. The exciting cause may be a sudden or a long-continued strain upon the emotions, or the muscles; a definite injury to nervous tissues, by traumatism, or the action of infectious diseases. Fright and anxiety have both been followed by it; it has followed prolonged night-watching, where the loss of sleep was made more detrimental by co-existent anxiety. It has been a result of anxiety and privation incident to war, both in our own country and abroad.

Occupations, involving the prolonged use of a limited number of muscles, are a frequent exciting cause. A prolonged, unusual strain upon a single set of muscles (carrying a heavy bag some miles was the cause of one case), has often been followed by the disease. Krafft-Ebing found that out of one hundred and ten cases, he could find seven where the disease had directly followed the injury of a particular nerve, and first appeared in the area of innervation of this nerve. It has closely succeeded illnesses from infectious causes, particularly after typhoid; rheumatism also; somewhat marked as the exciting causes in the unusual adolescent cases.

PATHOLOGY. It has been called a disease without a lesion, but this should be qualified by adding the adjective "definite," since changes have been found in all parts of the nervous system. Dana states that there is congestion and dilatation of the vessels in the gray matter, a diffuse increase of interstitial tissue, atrophy, and pigmentation of cells.

They are not constant, nor does their severity, or distribution give any criterion of the severity, or extent of the disease of which they might be supposed to be the cause. Autopsies have disclosed changes in the muscles similar to those in pseudo-hypertrophy; there is also a tendency to myxedematous overgrowth in the skin of the face, and in patches over the rest of the body; sclerotic changes have been disclosed in every part of the nervous system. On account of the changes in the skin and muscles, some have attributed it to a perversion, or lack, in some glandular secretion. Berkeley believed that the parathyroids were the glands concerned, and that the administration of parathyroid would cure the condition. Others, like Gowers, would localize it in the motor-centers of the cortex of the brain. Hughlings-Jackson considers that the disease is a form of hemiplegia, but it is pointed out that the lack of change in the reflexes, and the absence of ankle-clonus, make the position a hard one to understand. Taylor believes it to be based upon a disease of the caudate and lenticular nuclei, and not alone in the cells, but also in the association-tracts. This would ally it with chorea, and make both diseases depend upon an insufficiency of cerebral control over motor discharges.

Dana has suggested, that since it displays a retardation and an interruption in the execution of voluntary movements which demand smoothness and persistency of impulse, that it may be due to a lesion in the synapses (the structure made up of the terminal brush of the nerve-fibre and the dendrites of the cell, and the amorphous substance lying between them).

SYMPTOMATOLOGY. The invasion is generally slow, occupying from one to three years in attaining full developement, although it may be rapidly ushered in by an acute illness, or an attack of sciatica, or when caused by emotion. Tremor is the first obvious symptom, and it is initially seen in one hand, and then ascends the arm of that side, and next invades the leg. The same order is followed upon the other side, as a rule, but divergence from this order is not uncommon. Occasionally the invasion is abrupt, but usually it is a system of advance, and recession, and then advance, until the disease is fully localized. Long before any suspicion of the disease is excited, the person feels a weariness and stiffness of the muscles. This is seldom a paralysis at any stage, since a sharp command will provoke a prompt and adequate response. It arises possibly from the changes in the muscles, and causes a clumsiness in movement which becomes quite distinctive. It is not a spastic condition, since the reflexes are not exaggerated. This rigidity increases as the years pass, until the whole body is moved as if in one piece. If the patient wishes to change the direction of his vision, he may have to turn the whole body. While the respiratory muscles, and those of deglutition may feel stiff, the functions of both are accurately performed. The muscles of the face, being affected by the same rigidity, utterly lose their customary play of expression, and if, as is common, there is some muscular hypertrophy, and a thickening of the skin, the mask-like face is distinctive. The muscles of the eyelids, and eyeball, and the iris are all affected by this same weak rigidity, and their motions are clumsy and retarded. The fact that contracture ensues from the changes in the muscle structure, and that this contracture exercises its force toward flexion, produces conditions which are peculiar to the disease. The curvature of the body is forward, the arms drop to the sides, but with the elbows held out from the trunk, the forearms are bent at nearly a right-angle, and crossed over the chest or abdomen; the hands are bent inward on the forearms, and the fingers are bunched, and bent slightly toward the ulnar side. The forefinger and thumb are usually quite closely applied to one another, and this position combined with a nearly continuous fine tremor, produces the diagnostic "pill-rolling" movement. Another effect of this muscular condition is the characteristic gait. We make the ordinary movements in walking for a three-fold purpose, viz., to progress in a premeditated direction, to maintain our lateral balance, and to maintain our center of gravity in a fore-and-aft direction. At each minute of our progress we are making numerous delicate and instantaneous re-adjustments. In paralysis agitans the clumsy and weakened muscles fail in all these points, and so the gait becomes a trot, to catch up with the center of gravity, which has been displaced with the first step. Some persons cannot stop, and must run into some fixed object to bring them to a halt. This is called "festination." In some cases, if left alone, they will go indefinitely to one side; this

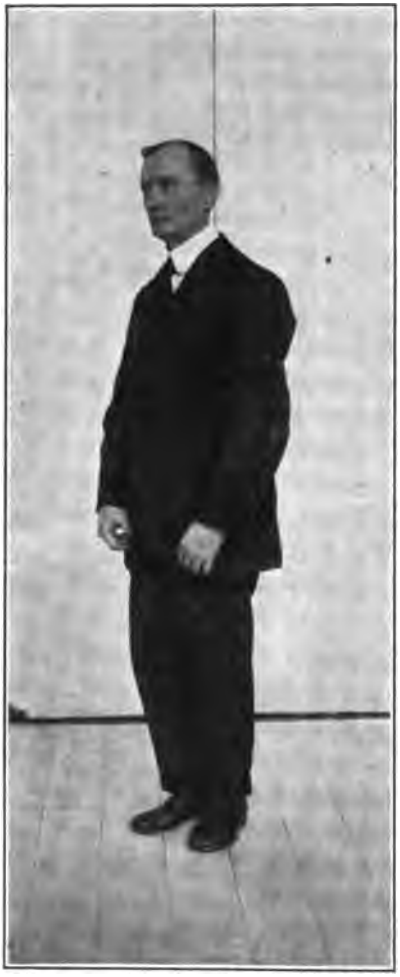


FIGURE 84. PARALYSIS AGITANS.
The right hand shows characteristic contraction.

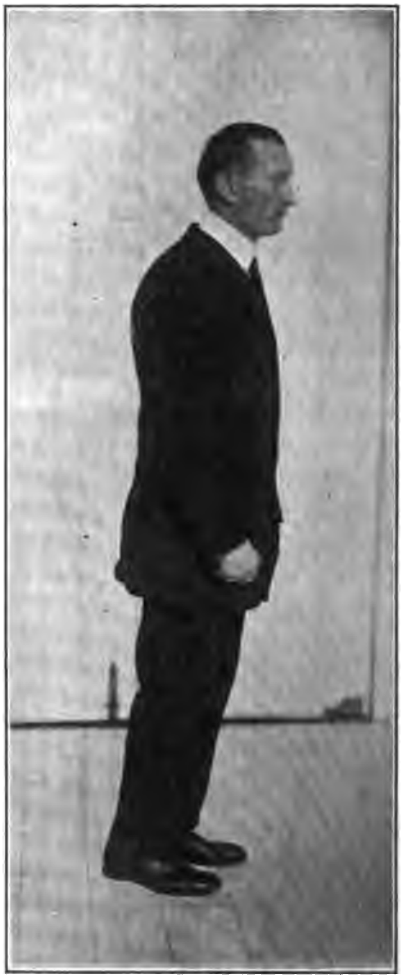


FIGURE 85. PARALYSIS AGITANS.
Attitude in walking, causing "Festination." Curving of the dorsal spine is also characteristic.

is called "latero-pulsion," and if to the rear, it is called "retropulsion," but this often occurs when they have been walking forward. In arising from a seat they push themselves up by the hands, and in seating themselves fall into, instead of sink into a seat. The contractures referred to as existing in the hands may also exist in the feet, and the flexion of the toes may prove a further embarrassment in locomotion. The rigidity and muscular changes may finally reach such a point that in the latter stages of the disease the patient may be confined to a chair or bed.

Tremor is the most characteristic symptom of the disease, and after it appears as a fixed symptom it is constant, never ceasing except in sleep, chloroform narcosis, and shortly before death. It is quite peripheral, affecting only the distal part of a limb. In a typical case it appears a few months after the muscular rigidity, but it is the first symptom in cases arising from shocks to the emotions, and is usually in the affected muscles, in those cases attributable to muscular strain, or the injury to a nerve. It is, however, absent in some traumatic cases. It is passive in type, i. e., while the parts are at rest; and disappears on volitional movements of the parts; it is coarse in extent, and slow in rate, i. e., between three and six to the second, usually about five oscillations to the second. In combination, these characteristics are distinctive. While it is passive as a rule, it is sometimes intention, i. e., increased by voluntary movement of the parts. The rate may be changed, and also the amplitude, by emotion or fatigue. It sometimes is a remittent symptom. It follows about the same rules in invasion and extension as do the weakness and rigidity, but it always spares the abdomen, the head, and the jaws. It may be so pronounced as to shake the whole body, giving rise to the idea that the head is also in tremor. Use of the arms in some cases will cause the feet to shake and drum upon the floor. When the head seems to be affected, if the patient is laid on a couch, so that the head and trunk are both supported, it will be found that the apparent tremor of the head was communicated to it by the violence of the shaking of the body. The tongue may show a fine tremor, but the lips are held firmly (tremor has been noted in one case); if the eyelids are closed they may disclose a fine tremor. Writing displays the tremor, as it is wavy, both on the up and down strokes. The muscles of phonation are weakened, and rigid, but do not display a tremor. The weakness and rigidity make the voice high-pitched and toneless, but the "jolting" phonation, sometimes evident, is caused by the general tremor of the body. The sphincters are not weakened, but, in some cases, micturition is hurried. The reflexes are not affected uniformly in all cases, or at different stages of the same case. In the beginning they are exaggerated, as in neurasthenia, and in late stages they are diminished as the rigidity becomes marked, but in no case are they abolished. Foot-clonus is never present. Electrical reactions are normal. The mental condition is unresponsive. It is frequently stated that they are unemotional, and live in a sort of "fool's paradise," but I have always found them easily moved to tears, and, less often, to laughter. They seem to be weak-minded, but this assumption is an error; the inertia, so evident in the muscular system, is equally true of mental processes, and a sudden strong stimulus will, in either case, provoke a normal response. They do not tend to terminate in dementia, but a small proportion of the cases mark the final stage by a mental breakdown, initiated by a rise in the body-temperature. Sensory changes are absent as a rule, but some cases have displayed a diminution in the perception of pain. This loss shaded off into the normal as the trunk was approached. Sometimes we find paresthesias, and, very rarely, a case may be neuralgic. Vasomotor changes are common. These manifest themselves in flushings; sensations of heat and cold, and in palpitation. The patients have attacks of sweating, and this is often localized, yet not always on the parts affected by tremor. The alteration of the secretory functions is also evinced by the increased flow of saliva, and the occurrence of diarrhea.

COURSE. The slow and remitting invasion has been described, and it may take years for the full picture to be developed. The gradual increase of all the symptoms finally reduces the patient to helplessness, but does not seem to materially shorten the expectation of life.

PROGNOSIS. The probability of cure is almost "nil," but the danger of an abbreviation of life is almost as slight. Even at the late period of life, when the majority are attacked, they have an expectation of life, all the way from ten to forty years. A few cures have been reported, but the author has seen only one case of amelioration even.

DIAGNOSIS. This rests upon the history that the patient had first a weariness, followed by a rigidity of the muscles; that he then developed a coarse, passive tremor of moderate rapidity, most marked in the extremities, and which spared the head and abdomen. That he had palpitation, flushings, and feelings of heat and cold. It depends upon the fact that the patient is bent and rigid; that he walks bowed over and with a trot; that his face and voice are both expressionless; that his pupils are sluggish, and his oculomotor muscles are rigid; that he is mentally unemotional. That with all this, his reflexes are not organically disturbed and that his electrical reactions are normal. Incomplete cases must be diagnosed on history and symptoms present, taking into account their order of evolution, giving due weight to heredity, and to the age, and occupation, and habits of the patient.

DIFFERENTIAL DIAGNOSIS. Multiple Sclerosis. This disease is most often confused with paralysis agitans because both have tremor, but multiple sclerosis is a disease of those under thirty, while paralysis agitans very rarely affects those under forty. Objectively also; the paralysis agitans patient is contracted in flexure, while multiple sclerosis has no flexure, but has a foot-clonus, staccato-speech, and nystagmus, which are never found in paralysis agitans. The tremor is also different. It is passive and continuous in paralysis agitans, intention only in multiple sclerosis; it affects the head in multiple sclerosis, never in paralysis agitans. Ask the paralysis agitans patient to perform an act, and the tremor fades away as he approaches the goal, while initially the multiple sclerosis patient may display none, but on attempting the desired movement it becomes evident, and grows wilder and more active as he approaches the consummation of his task. In the rare cases where paralysis agitans has a tremor of the intention type, the absence of organically disturbed reflexes, and changes in the optic nerve, will bar the assumption that the disease is multiple sclerosis. The rigidities also differ. Multiple sclerosis has spastic rigidity, i. e., from a sclerosis of the lateral tracts, while that of paralysis agitans is from a change in the muscles (although we do not certainly know how, or whence, it is inaugurated). The first is a spasticity, while the second is a rigidity. If pressure is exerted upon a spastic limb, it withstands it for a few seconds, and then suddenly gives way ("lead-pipe" rigidity); put pressure upon a rigid limb and it bends at once, up to a certain point, and never gives way beyond that point.

Simple Tremor. This begins early in life, varies little as the years pass, and markedly affects the head, and often that only, and is often directly hereditary.

Senile Tremor. This mostly affects the head; usually primary there; fine in time and extent. At first it is intention, but later is passive; it does not tend to increase. It is sometimes very difficult to diagnose from the appearance of the tremor, but history may be an aid. The tremor of paralysis agitans begins upon one side, while that of age upon both, simultaneously. Remember that the two conditions may co-exist.

General Paresis. This has a fine tremor, and differs in several particulars. It is not passive, but evident only when muscles are put into a state of tension. It is then particularly evident in the face and lips. The personal history is different; the reflexes tend to be abolished; the mental state is not apathetic; but is marked by a deep melancholy, or of great exaltation. Lymphocytosis of the cerebro-spinal fluid in paresis will certainly differentiate.

Post-hemiplegic Tremor. It has been said that the tremor of paralysis might be hemiplegic, or initially so: this is true of post-hemiplegic tremor.

This latter, however, has the history of a preceding apoplexy; if that cannot be obtained, we can find some actual paralysis; there will be an exaggeration of the deep reflexes.

Toxic Tremors. Sensory symptoms are common in all of these, and on the other hand, all toxemias may be barred if we find a fixed attitude, immobile face, and festination. Alcohol produces an irregular tremor which is wide in range, and is evident in the legs when the patient attempts to stand. The individual is in a state of constant restlessness. He has foot-drop and gastritis, and his nerve-trunks are very sore. The tremor of mercury is often the first symptom to appear and it is very coarse. It is all over, and is intention, but may be constant. It shows first in the face and tongue. The patient has a stomatitis, and an irritable stomach. Arsenic shows pain before the tremor appears. While lead has a marked tremor, it usually can be detected by a wrist-drop without pain, with much motor-weakness, and the blue line on the gums.

Traumatic Neurasthenia. This may at times produce a similar tremor, but the period of invasion is very much shorter, and as time goes on, rigidities are hardly ever developed in the neurasthenic case, and the peculiar gait never. The anemic state is similar, but it is amenable to treatment only in the neurasthenic case.

Hysteria. This may have a like tremor, and similar bodily conditions, but the hysterical patient (except in traumatic cases) is a young female, who has stigmata, particularly restricted visual fields, and contracted, or reversed color-fields. The tremor, while similar, is almost entirely controlled by psychic stimuli. The mental condition is never so placid as in paralysis agitans, and the inception of the case was from a class of excitants which differs from those of paralysis agitans.

Arthritis Deformans. Literature furnishes one case of this disease which presented a tremor of the proper type, and the distinctive attitude, but as this case had also a cervico-dorsal kyphosis the writer questioned whether the tremor might not have been produced by pressure upon the motor cervical roots.

Senile Paraplegia. This depends upon an arterio-sclerosis of the brain and spinal cord. These cases exhibit the posture and tremor of paralysis agitans, but they will also develope some paralyses, and defects in speech, swallowing, urination, etc.

TREATMENT. In some cases improvement has followed the administration of remedies, but the possibility of cure is denied by those whose wide experience, and judicial temper of mind entitles them to speak with authority.

Under such conditions we are not justified in urging treatment upon those afflicted with the disease, unless we are convinced that it will stay the disease to some extent, or that it will render them more comfortable in body or mind. Experience has demonstrated that treatment does mitigate many accessory symptoms, the anemia common to all these cases can be benefitted, and the patient is rendered much happier by the knowledge that some attempts are being made to mitigate his condition. Upon these grounds the patient should be urged to live a quiet life, as free as possible from physical strain and worry; he should be as much as possible in the open air, but absolute rest is contraindicated. A plain diet should be enjoined, although some alcohol is often a benefit. He should choose an equable climate to reduce the strain of life, although no climate is in any degree curative. The advantages of exercise without fatigue may be obtained from massage, but it must be adapted to the specific case, since some persons are appreciably irritated by routine treatment, and frequent rubbing with the hand may be more beneficial. Persons have thought that in the early stages benefit has been derived from suspension in a Sayre's apparatus. Some patients seem better after a carriage ride, attributing this benefit to the jolting. On this account Charcot advised the use of a jolting-chair, or some other method of vibration. Gowers says that this is contrary to common experience. Westpfal advised nerve-stretching, but this has been generally condemned. Cold baths are harmful, and while hot baths are advised by some, the general opinion seems to incline toward avoidance of all extremes of temperature. Lukewarm baths of twenty minutes' duration night and morning relieve the vasomotor symptoms, and reduce the tremor in some cases. These should be combined with electricity and massage. This brings up the question of the value of electricity. It is not considered to be of much curative value, but since many patients will have it, from a charlatan if not from their own physician, its utility must be considered. King concludes as follows: "Electricity is not essential for the purpose of nourishing the muscles, but may be used by passing the stabile galvanic, or other current, through the muscles. Neither an interrupted galvanic or a faradic is ever indicated. Some results have recently been secured by central galvanism. Little good can be expected in chronic cases, although painful sensations in rigid muscles, insomnia, and anorexia, might be relieved by proper electrical applications." Tremor, and uneasiness of muscles may also be relieved to some extent. Charcot recommends the static breeze with an occasional discharge of sparks, which he states to act particularly well upon the tremor. Laret reports a single case which showed a marked amelioration of the symptoms from the use of the high-frequency current, in connection with the static spark. He also finds that vibratory treatment in connection with static electricity is palliative in many cases. Eulenberg advises a weak current through the head, and Beard and Rockwell galvanization of the sympathetic nerves and the brain. Gray used a large flat electrode to the nape of the neck, and another somewhat smaller, to the lumbar spine. Current of from three to five m.a. increased later to fifteen m.a. for five minutes, three times a week. It is doubtful if the spinal cord can be influenced by electricity applied in such a manner.

R. Friedlander applies gymnastics and Swedish movements to the relief of the condition. He makes the patient conquer the tremor by directing the attention to the small muscular groups, and later to the larger. As the flexors are most affected, he endeavors to strengthen the extensors by resistive exercises, and so he expects to gradually conquer the rigidity. A natural swing to the arm and leg is next attempted. He makes a slow heel-and-toe-gait, of varying length, an exercise, until the patient can perform it without mental effort. He makes much of his "falling exercise," in which a certain part of the body is raised passively, and then allowed to fall, the patient endeavoring to keep the part entirely relaxed at all times. He believes that by this exercise the patient's power of voluntary relaxation is much increased. The guide to the amount of exertion is the pulse rate. Upon any acceleration the exercise must be stopped until it again becomes normal. Several weeks are needed for a sufficient trial of this method.

THERAPEUTICS. We can hope to accomplish very little with remedies, and many of the remedies advised in our text-books, while they have tremor and stiffness of the muscles in their pathogenesis, will be found to be inapplicable, since these will be found to have been spasticities instead of rigidities. Argentum nit. has clinical evidence of some potency in this disease. Its symptomatology comprises the tremor, muscular weakness and rigidity, flushing and palpitation. Baryta carb. and mur. should be thought of in these cases on account of their efficiency in changes which suggest senility, or pre-senility. Where tremor is the initial symptom we should think of Agaricus or Agaracin in the 2x. Gelsemium has the tremor especially marked, but, at the best, it can be considered a remedy for the initial stages only, and only then when the vasomotor symptoms are a deficiency, instead of the usual increase in arterial tension. Hyoscyamus has tremor and stiffness of the limbs, and peculiarly the flexor spasm of the toes. It may be of some service. Sparteine 1x to 2x, was given to a patient for the weak condition of the heart, and unexpectedly, the tremor was greatly improved. Physostigma has been given for cases having a clear sensorium, and a tremor apparently arising from a circulatory disorder of the spine. The following have also been recommended: Arsenicum, Causticum, Ergot, Ignatia, Lycopodium, Mercurius (which has been advised on the ground that it will benefit any interstitial overgrowth of the nervous system. The author saw very considerable improvement in a case of long standing following its use), Oleum Jecoris asseli, Phosphoric acid, Plumbum (strongly endorsed as at least palliative of the symptoms), Rhus (when the patient has been rheumatic and the invasion was subsequent to exposure to cold and wet), Stramonium, Scutellaria, Zinc, and the Bromide of Zinc.

Dewey says that Plumbum, Atropia sulph., and Hyoscyamus are the remedies for paralysis agitans.

Physiological Therapeutics. The general acceptation of an intoxication theory of the disease has led to experimentation with the glandular extracts and calcium salts. Thyroids have not proved to exercise any beneficial effect, while some lessening of the tremor has been observed after the use of an extract of hypophysis, with more quiet sleep, and an improvement of the mental condition. Berkeley, of this city, has used a preparation of the parathyroid in 65 cases with improvement in many, and apparent cure in some of them. Others have reported distinct improvement in as many as one-third in a group of cases, and Jelliffe reports the reduction of the tremor to a remarkable degree in one case, and distinct improvement in many others. The gland is prepared in tablets of 1/10 grain, and three a day are administered.

Hyoscine hydrobromate is the drug of most common use. It is given for the reduction of the tremor. It is the most efficient drug for the reduction of the tremor, and also for the relaxation of the muscles. The relief is not permanent, its use may give rise to unpleasant symptoms, and its withdrawal after long use may induce symptoms of collapse. It is given hypodermically, and by the mouth, and Starr advises doses of 1/200 of a grain every six hours, then more frequently until the tremor is quieted, and then three doses a day may be sufficient. He finds that the addition of 1/6 grain of codeine assists its action while Jelliffe adds Sodium Bromide.

Duboisin is given as an alternate remedy; it is believed to be as efficient and less toxic. The dose is 1/100 grain, and is advised when Hyoscine fails to relieve.

Arsenic is prescribed for the anemia, since it has no effect upon the tremor or the rigidity. Given as the Sodium Cacodylate, or Fowler's solution, in doses of 2-3 drops three times a day.

Amyl nitrite has been given for the vascular symptoms; but never Digitalis.

Atropine will check the tremor in some cases, but if given in sufficient doses it dries the mouth too much to be tolerated. This effect is seen to some extent in the use of Hyoscine.

Cannabis Indica is given in pills of ¼ grain (Starr). Tincture of Veratrum viride 3-5 drops three times a day (Oppenheim), and Tincture of Gelsemium have also been advised.

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